JUN 18 1924

VOLUME 27 NUMBER 6

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

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JUNE, 1924

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $4.00

Entered as Second-Class Matter, January 4, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

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American Journal of Diseases of Children

VOL. 27 JUNE, 1924 NO. 6

STUDIES IN GROWTH

I. GROWTH OF NORMAL CHILDREN *

FRITZ B. TALBOT, M.D.

BOSTON

The need for normal figures on the growth of different parts of the body presented itself to me as a result of my studies on the growth and metabolism in pathologic cases during the last two years.[1] Since these data were not available, figures were compiled from a series of body surface measurements, taken, according to the Du Bois linear formula, on a group of clinically normal American children on whom studies had previously been made to determine the normal standards of basal metabolism.[2] These were average children within the accepted normal limits of height and weight for age, and do not represent exceptionally well nourished or poorly developed children. A description of these children has been given in an earlier publication.[2]

The measurements were plotted on charts, and smoothed curves drawn to indicate the trend of growth of the different parts of the body. These curves do not represent mathematical averages. The distribution of the dots on either side of the curve show the normal deviation from the average, which must be taken into consideration in all instances.

In estimating growth the weight to age and height to age ratio are most commonly used. The height and weight curves of this series are presented in Charts 1 and 2. These measurements were taken without clothing.

The curves of growth in height and weight of this series show a close uniformity to the Crum-Wood curve, but are lower than the curves of private school children measured for Holt in New York [3] and Benedict and Talbot in Boston.[2] This is owing to the fact that this series represents a group of average children, while the private school group represents children living in exceptionally good surroundings.

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* Received for publication, March 24, 1924.

* From Children's Medical Department, Massachusetts General Hospital.

1. Talbot, F. B.: Tr. Am. Ped. Soc. **35**:322, 1923.

2. Benedict and Talbot: Carnegie Ins. Wash. Publication No. 302, 1921.

3. Holt, L. E.: Standards for Growth and Nutrition, Am. J. Dis. Child. **16**: 359 (Dec.) 1918.

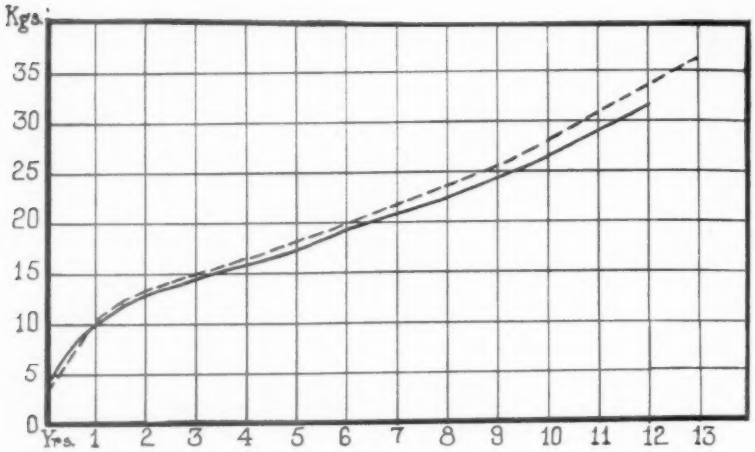

Chart 1.—Weight referred to age. In these charts, the broken line represents boys; the straight line, girls.[2]

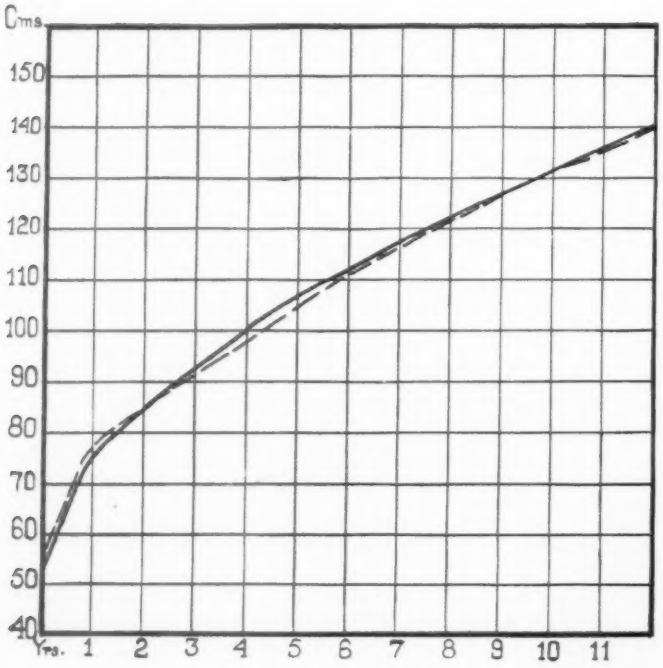

Chart 2.—Height referred to age.[2]

In order to obtain accurate data concerning relative body proportions, the children should be grouped according to weight for height which admits of less variability than age. Measurements of growth from the standpoint of stature are presented in Chart 3.

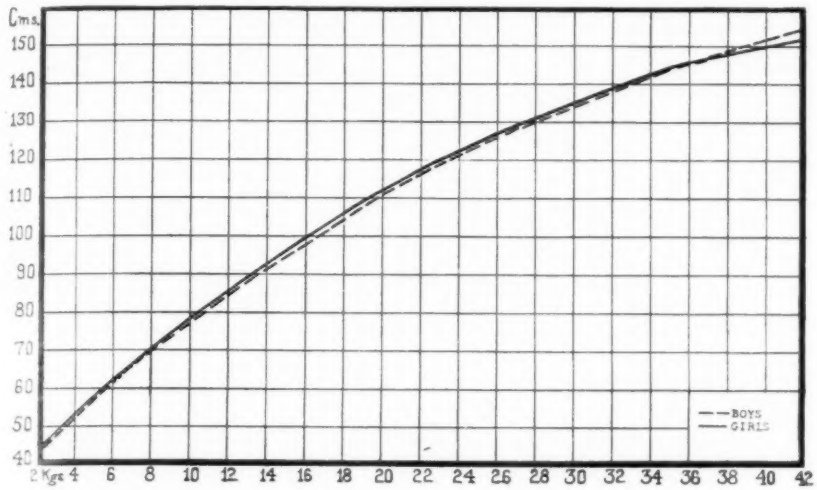

Chart 3.—Height referred to body weight.[2]

Circumference of Head.—The measurements of the development of body proportions are based on growth according to age. Charts 4 and 5 show the growth curve of the circumference of the head of boys and girls. This was measured from the occiput around the frontal bosses. Each dot represents the measurement of a child at a given age.

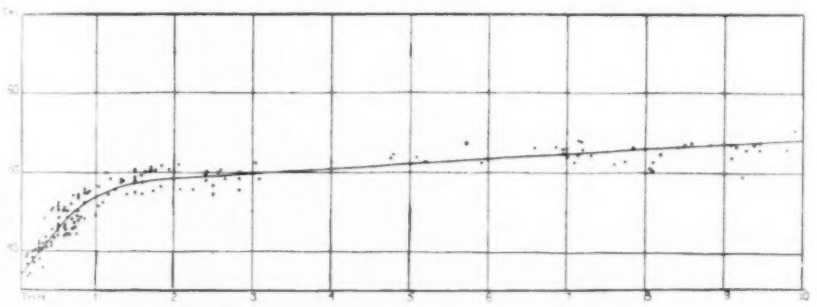

Chart 4.—The circumference of boys' heads.

The circumference of the head of boys is somewhat larger than that of girls. The very rapid increase in the size of the head during the first year of life in both sexes is followed by a gradual regular increase up to the age of 10 years. This great increase in the circumference of the head during the first year corresponds with the very rapid growth of

the brain, which is said to double its weight during the first 8 months of life.[4]

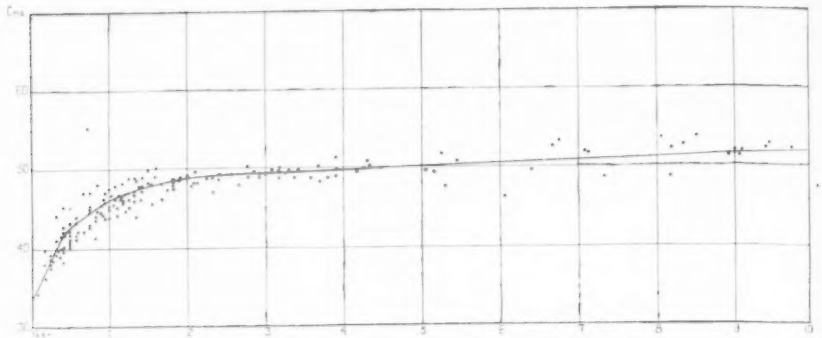

Chart 5.—The circumference of girls' heads.

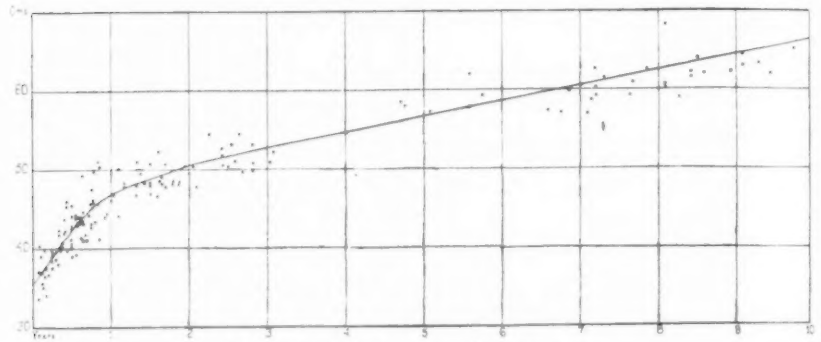

Chart 6.—Chest circumference, boys.

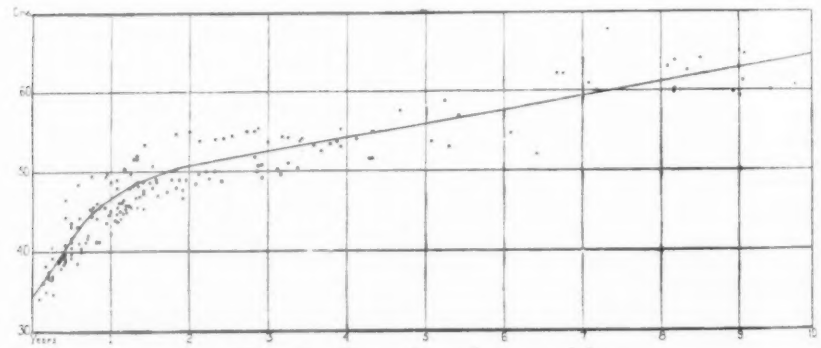

Chart 7.—Chest circumference, girls.

Circumference of Chest.—Measurements of the circumference of the chest (Charts 6 and 7), were taken over the nipples half way between full inspiration and expiration.

4. Gundobin: Die Besonderheiten des Kindesalters, Berlin, 1912.

A close correlation is seen in the rate of growth of the chest in both sexes. The circumference of the head and chest is almost identical until 18 months of age, after which the circumference of the chest increases much more rapidly than that of the head.

Circumference of Abdomen.—Measurements of the circumference of the abdomen were taken over the umbilicus. There is a fairly close uniformity in the trend of the curve in both sexes. The great diversity in the distribution of the dots, especially in infancy, is explicable by the fact that a recent meal or gas may distend the abdomen and thus increase its circumference.

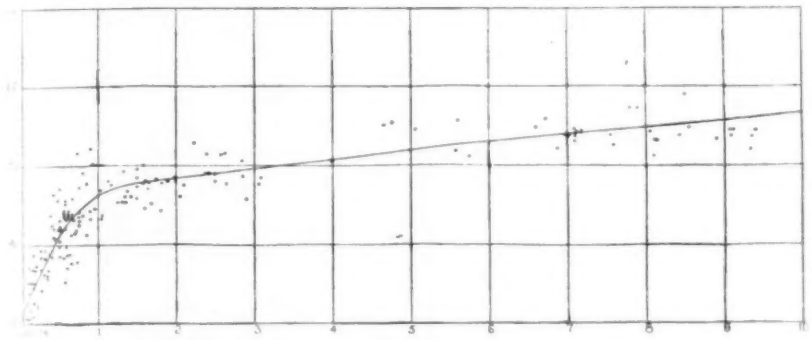

Chart 8.—Circumference of abdomen of boys.

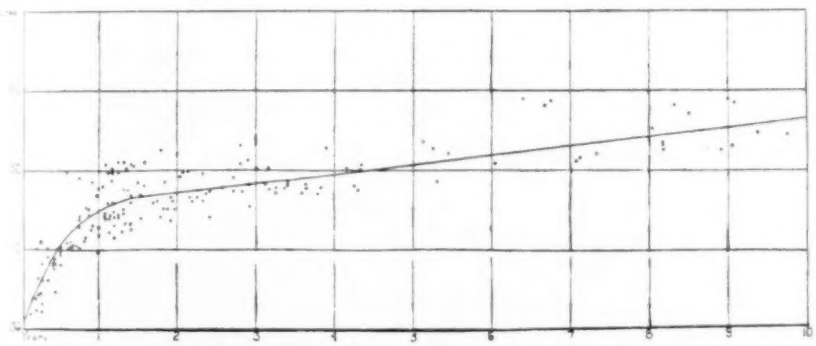

Chart 9.—Circumference of abdomen of girls.

Length of Trunk.—Measurements of the trunk were taken from the upper end of the sternum to the top of the pubis. Such measurements are very difficult to obtain in early infancy and should only be carried out with the infant lying flat on his back, on a smooth surface, in order to insure a perfectly straight spine and thus avoid errors due to flexion or extension.

These curves show a close uniformity in the rate of growth of the trunk in both sexes.

Length of Legs.—Leg measurements of the children are seen in Charts 12 and 13. These measurements were taken from the top of the pubis in the mid-line to the bottom of the foot.

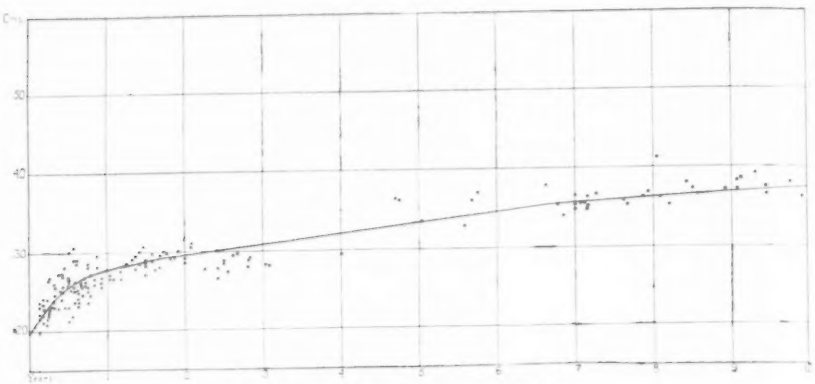

Chart 10.—Length of trunk, boys.

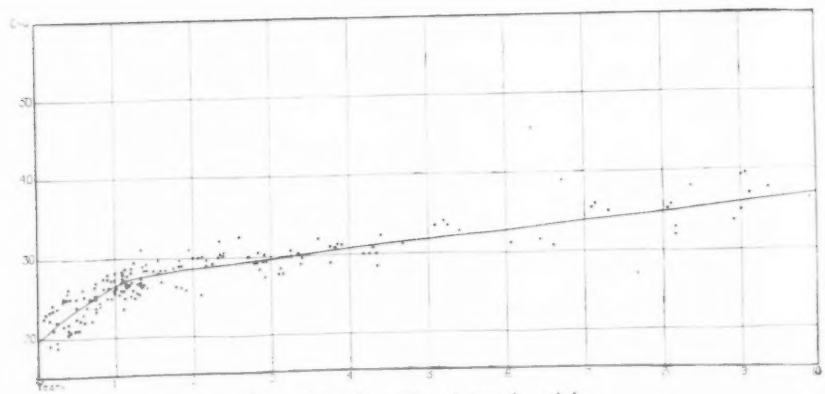

Chart 11.—Length of trunk, girls.

There is very little variation in the length of the leg of the two sexes up to the age of 6 years, at which time the rate of growth is greater in boys than in girls. The relatively greater growth of the legs than trunk is in conformity with the transition from the infantile proportions, in which the legs form three eighths of the total height, to that of the adult, in which they are about one-half of the total height.

Length of Foot.—Measurements of the length of the foot were taken from the heel to the tip of the great toe. There is very little if any difference in the length of the foot of boys or girls up to the age presented.

Total Length of Arm and Hand.—This was measured from the tip of the acromion process to the end of the second finger. Differences are more apparent in the length of the arm of boys and girls. From 2 to 6 years there is a greater length of arm in girls; from 6 years on it is greater in boys.

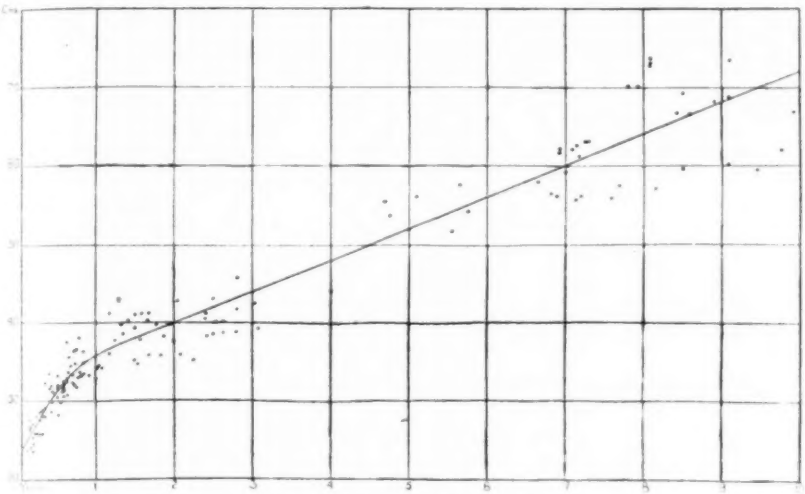

Chart 12.—Length of leg, boys.

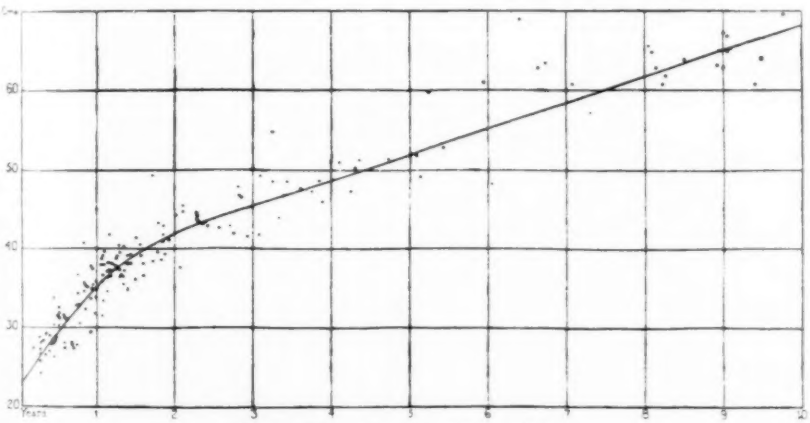

Chart 13.—Length of leg, girls.

COMMENT

These charts on the development of body proportions show very little sexual differentiation. A study of the curves leads to the conclusion that there is an "expected growth" for a normal individual. Measure-

ments of normal children may either be applied to the curves at intervals and their relative physical development estimated, or comparison of single observations may be made in order to show their relation to the normal.

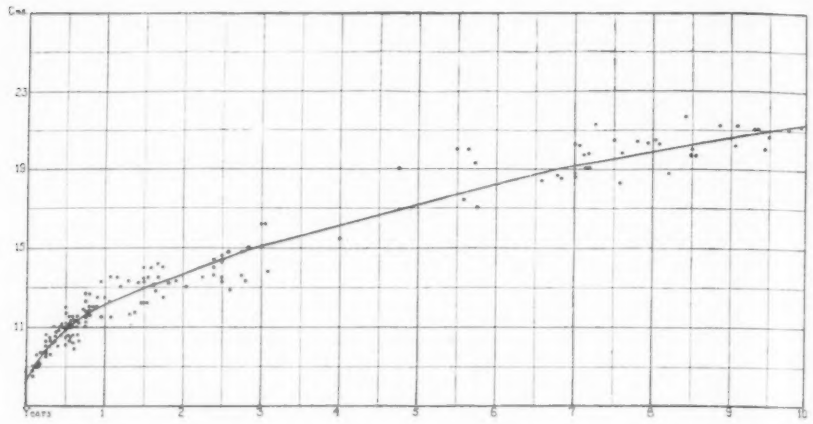

Chart 14.—Length of foot, boys.

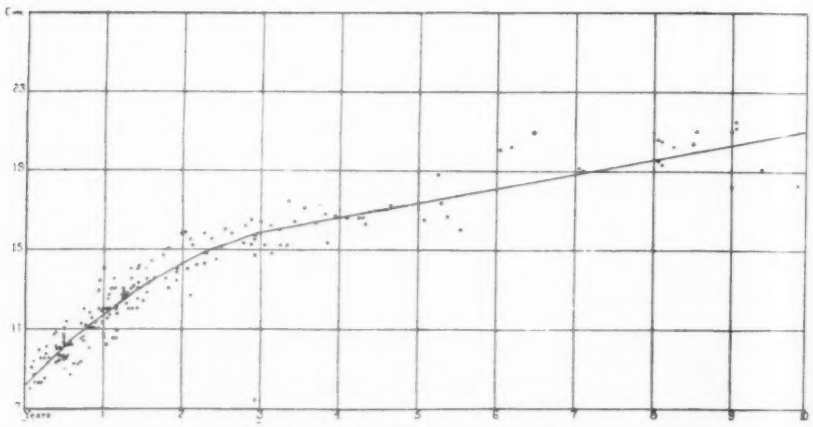

Chart 15.—Length of foot, girls.

Abnormal cases may be compared with these curves to determine in what respect their physical development differs from the normal. This has been done with cretins,[1] mongolian idiots and premature infants.[5]

The effect of treatment on the growth of abnormal children may also be studied by comparing them with these curves. If the growth curve runs parallel with or falls away from the normal curve, treatment has

5. To be published.

not affected growth. If, on the other hand, the growth curve approaches or reaches the normal curve, treatment may be said to have stimulated growth.

The interpretation of results of basal metabolism studies in childhood is very difficult, principally because of the differences due to physical development; these differences becoming more marked as the child

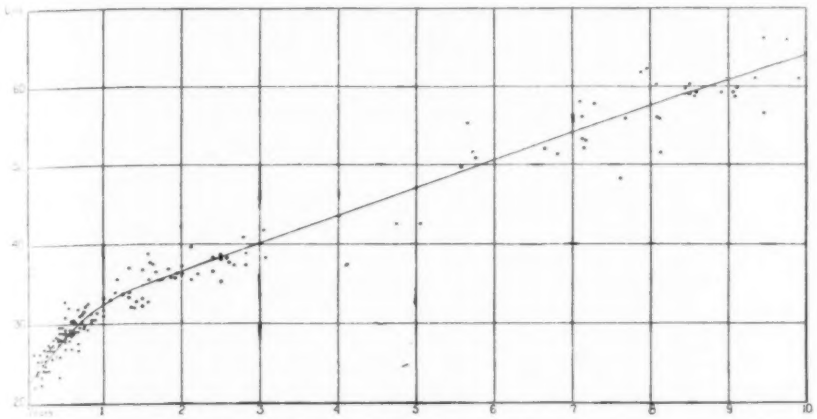

Chart 16.—Length of arms, boys.

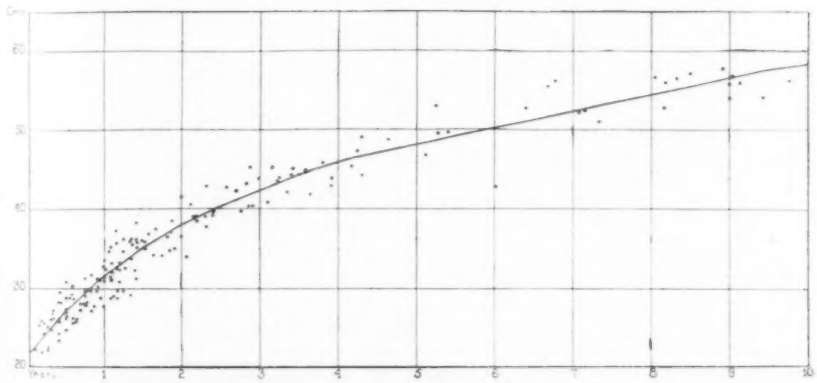

Chart 17.—Length of arms, girls.

approaches puberty. It is, therefore, very important to estimate the physical development of a child whose basal metabolism is to be determined. This may be done by comparing the body measurements with the foregoing curves of growth of children of the same age.

If the development is found to be abnormal, the basal metabolism should then be compared with that of children of the same physical

development irrespective of age. This is seen in cases of malnutrition and undernourishment which are accompanied by variations in the basal metabolism.[6]

SUMMARY

Growth curves of clinically normal American children are presented. They may be used as a means of comparison in the study of growth of untreated cretins, mongolian idiots and other abnormalities of growth, and are of value in determining the physical status of a child whose basal metabolism has been studied.

Body Measurements of Premature Babies

						Length				Circumference			Estimated
Case	Sex*	Age, Days	Wt., Kg.	Ht., Cm.	Sitting Ht., Cm.	Trunk, Cm.	Arm, Cm.	Leg, Cm.	Foot, Cm.	Head, Cm.	Chest, Cm.	Abdomen, Cm.	Prematurity Days
25	♀	1	0.727	34.5	23.3	12.8	14.4	14.2	4.9	23.1	20.3	16.9	—90‡
11	♂	18	0.992	....	....	15.9	16.9	14.9	6.03	27.6	15.2	20.3	—75
		38	1.191	38.0	....	13.3	16.1	16.2	6.1	28.0	26.0	19.7	—55
22	♀	7	1.304	38.0	25.0	12.5	17.2	14.3	6.0	25.2	24.0	24.0	....
1	♀	8	1.361	42.0	28.0	14.3	17.4	16.5	6.0	28.7	23.0	21.8	—60
19	♂	28	1.460	44.5	....	15.8	17.8	17.9	6.4	29.9	22.9	22.5	—30
3	♂	18	1.474	42.5	26.5	17.0	17.3	17.5	6.5	30.2	25.0	23.2	....
		34	1.843	42.5	29.1	15.1	17.7	18.1	7.0	31.2	25.0	24.0	....
13	♂	51	1.58	40.5	....	15.0	16.4	18.8	6.0	29.2	28.3	25.0	— 9
2	♂	18	1.644	46.0	28.0	15.0	19.8	18.3	6.7	31.0	27.5	26.5	....
		34	2.155	47.0	30.5	15.7	20.3	18.0	7.2	31.9	27.7	30.1	....
7	♀	21	1.687	47.0	29.5	14.2	16.4	16.4	6.0	30.0	25.0	21.0	—30
		65	2.467	47.1	35.4	19.1	17.9	19.9	7.0	35.2	26.2	26.5	— 5
9	♀	3	1.701	43.5	28.5	13.0	17.9	17.6	6.5	31.5	24.8	22.8	....
		24	1.900	45.5	29.1	15.1	18.1	19.0	7.1	31.5	27.2	25.4	....
8	♀	23	1.723	45.0	30.5	17.3	18.3	18.7	6.1	29.5	26.8	29.0	—37
5	♀	12	1.744	43.0	27.4	15.1	18.9	18.7	6.6	29.3	25.5	23.0	....
		25	1.970	46.8	29.2	14.5	17.6	18.0	7.2	30.0	26.0	23.3	....
10	♂	7	1.744	43.0	26.2	14.9	16.4	16.3	6.6	29.8	24.5	24.0	—44
4	♂	12	1.76	42.5	....	14.0	19.0	17.9	6.7	30.0	25.0	23.0	....
		24	1.829	45.5	28.5	14.0	19.0	18.9	7.0	30.5	27.8	28.0	....
6	♀	14	1.772	43.8	29.0	16.2	15.5	17.5	6.1	29.7	26.7	25.3	—39
		68	2.86	47.0	....	16.1	16.0	19.1	7.3	33.4	31.1	30.6	+19
		97	3.02	53.0	34.0	19.5	20.5	20.4	7.6	34.5	33.0	33.7	+46
		14 mo.	6.861	68.7	47.2	24.1	28.6	29.9	10.8	44.3	41.2	40.0	....
		17 mo.	7.825	69.8	48.2	26.6	30.4	32.4	10.4	43.7	43.8	43.5	....
18	♂	21 da.	1.895	46.1	....	16.9	18.0	17.5	7.2	32.2	25.7	26.0	....
17	♂	28 da.	1.999	45.8	....	18.8	18.8	18.4	6.2	31.9	26.2	25.3	—40
23	♀	16 da.	2.098	46.5	....	16.2	19.6	20.3	7.8	30.6	36.1	24.4	....
15	♀	45 da.	2.155	45.5	....	19.0	18.1	19.1	7.0	32.3	28.9	27.5	....
16	♀	45 da.	2.339	47.7	....	21.0	17.7	21.0	7.1	32.4	29.0	27.8	....
24	♂	16 mo.	10.9	81.0	....	26.7	30.2	35.0	11.5	49.0	50.0	53.0	....

* In this column, ♀ indicates female; ♂, male.
‡ Measurements taken at necropsy.

II. GROWTH OF PREMATURE INFANTS

The curves showing the growth of normal children have been presented. These curves were constructed from measurements taken according to the Du Bois linear formula.[7] The following measurements of a series of premature infants, on many of whom basal metabolism

6. Talbot, F. B.: Severe Infantile Malnutrition, Am. J. Dis. Child. **22**:358 (Oct.) 1921.

7. DuBois, Delafield, and DuBois, E. F.: The Measurement of the Surface Area of Man, Arch. Int. Med. **15**:868 (May) 1915.

studies have already been reported,[8] were taken in the same manner and are presented in the accompanying charts for comparison with the growth curves of the normal series.

Individual measurements of these premature infants are presented in the table.

The measurements of these premature infants, of varying ages postpartum, are shown in the following charts in relation to the normal curve. Since it was difficult to estimate accurately the amount of prematurity, they are compared with infants of the same age after birth.

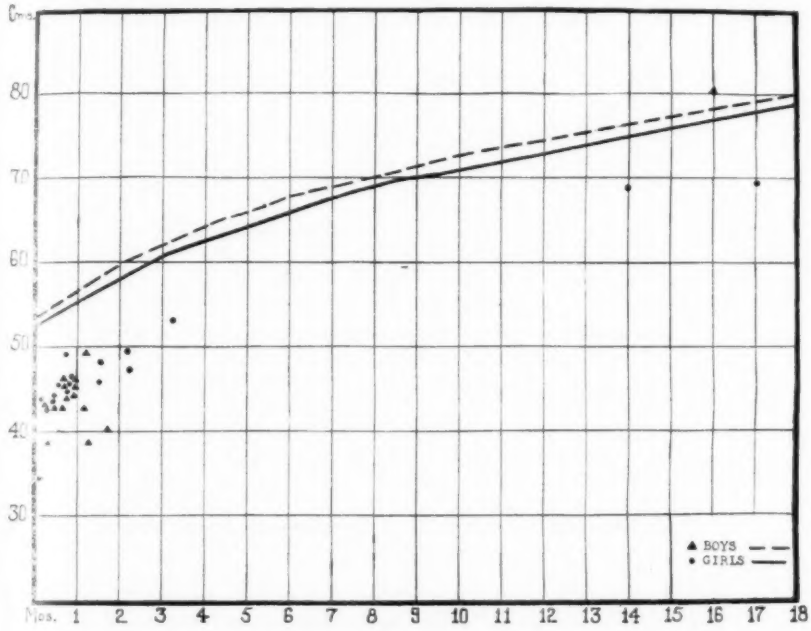

Chart 18.—Height referred to age in premature infants.

If it had been possible to measure the exact degree of prematurity, it would have been instructive to compare their development with the fetus of the same age.

Chart 19 shows that there is approximately the same relationship between the height and weight of premature and normal infants. The projection of the curve of normal children to the left would approximately bisect the dots of the premature infants.

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8. Talbot, F. B., and Sisson, W. R.: Proc. Soc. Exper. Biol. & Med. **19:** 309, 1922; Talbot, et al.: The Basal Metabolism of Prematurity, Am. J. Dis. Child. **24:**95 (Aug.) 1922; The Basal Metabolism of Prematurity. III. Metabolism Findings in Twenty-One Premature Infants, ibid. **26:**29 (July) 1923.

In the plotting of the curve and the dots no sexual differentiation has been made.

Length of Trunk.—Measurements of the trunk were taken from the upper end of the sternum to the top of the pubis.

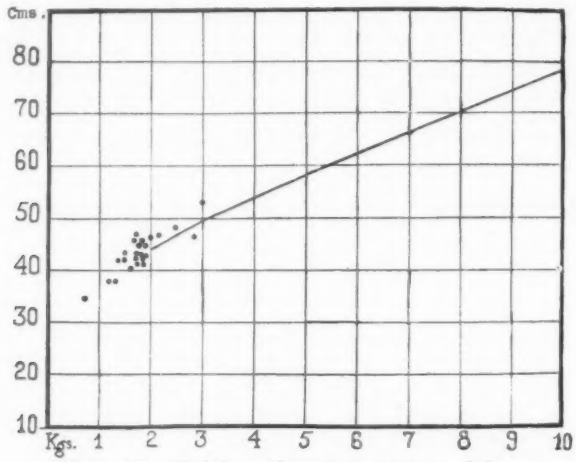

Chart 19.—Height referred to body weight.

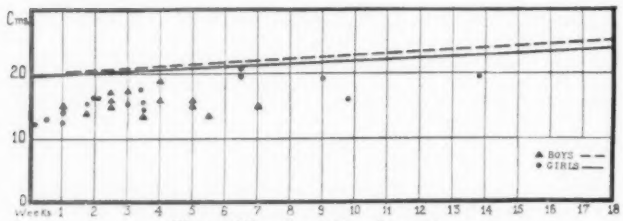

Chart 20.—Length of trunk.

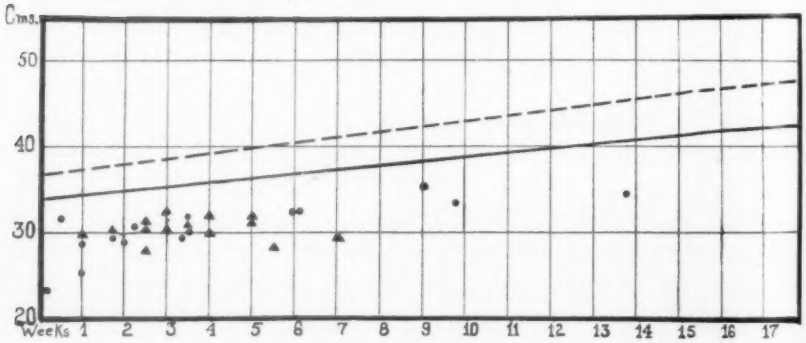

Chart 21.—Circumference of head.

Circumference of Head.—Measurements of the circumference of the head were taken over the occiput around the frontal bosses.

Circumference of Chest.—Measurements of the circumference of the chest were taken over the nipples, half way between full inspiration and expiration.

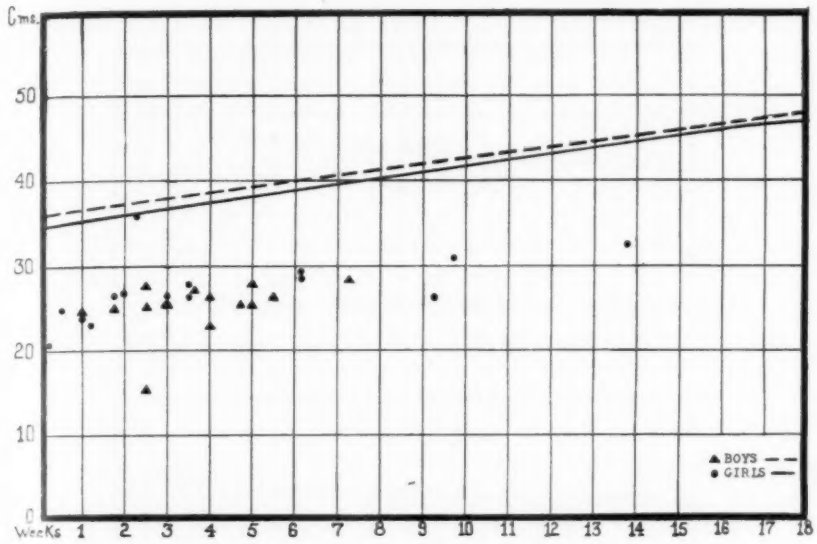

Chart 22.—Circumference of chest.

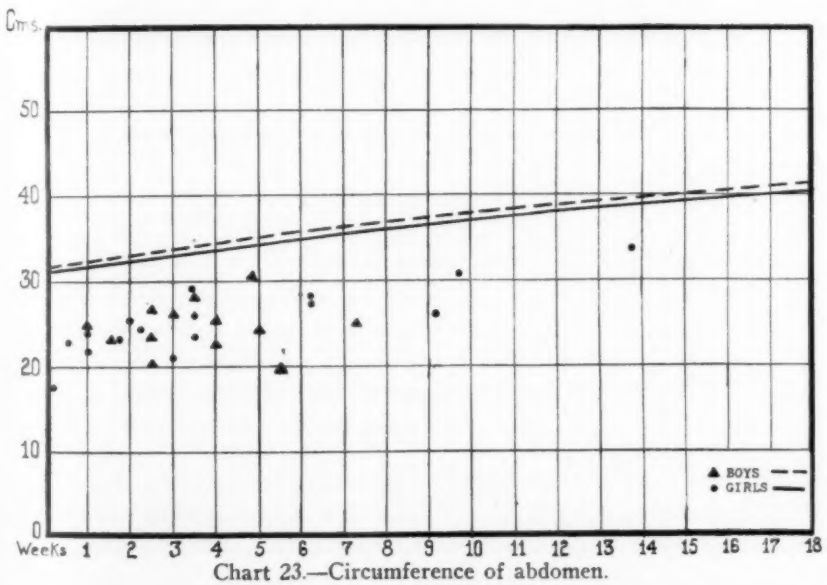

Chart 23.—Circumference of abdomen.

Circumference of Abdomen.—Measurements of the circumference of the abdomen were taken over the umbilicus.

Chart 21 shows that the circumference of the head of premature infants is closer to the normal than that of the chest or abdomen. This relation persists for several weeks after birth, and agrees with Ylppö's [9] findings that at birth the circumference of the head of premature infants

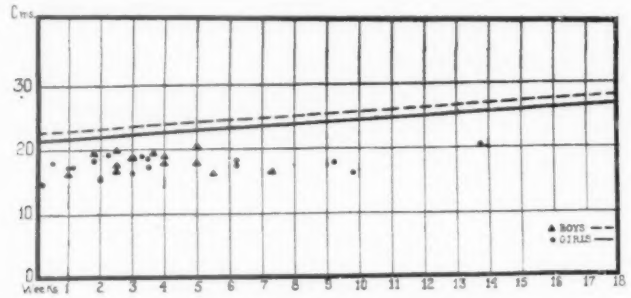

Chart 24.—Length of arms and hands.

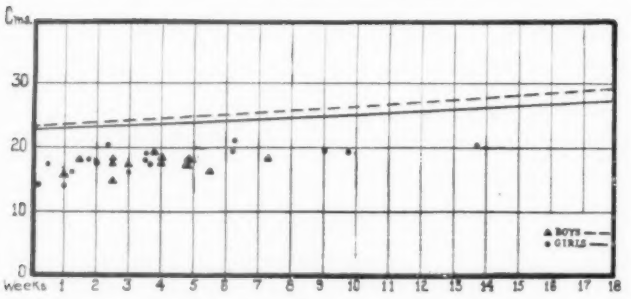

Chart 25.—Length of legs.

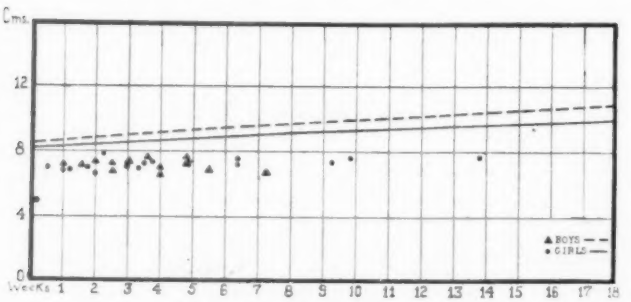

Chart 26.—Length of foot.

is greater than that of the chest, while in full-term infants the circumferences are similar. He also showed that the greater the prematurity the more marked is the disproportion between the circumference of the head and chest. It has been difficult to confirm this in our series because of the uncertainty of the time of expected birth.

9. Ylppö: Ztschr. f. Kinderh. **24**:110, 1919.

Length of Arms and Hands.—Measurements of the arms and hands were taken from the tip of the acromion process to the end of the second finger.

Length of Legs.—Measurements of the length of the legs were taken from the top of the pubis in the mid-line to the bottom of the foot.

Length of Foot.—Measurements of the length of the foot were taken from the heel to the tip of the great toe.

The outstanding feature in the comparison of body measurements of premature infants with normal infants is that the premature infant is smaller at birth in all respects. This condition persists for many months after birth. No figures are available to show when the premature infant enters the normal zone. A premature infant, seen in private practice, who had been fed on breast milk, and was living in the most hygienic surroundings, was measured at 16 months and found to be within the normal limits. Another infant, seen in hospital practice, living in relatively poor surroundings, and fed with breast milk only for the first few weeks, had not reached normal at 17 months. In the opinion of many pediatricians, it takes a premature infant from 1 to 6 years to reach normal. It seems probable that much depends on how premature the infant is, whether it receives breast milk or not, and whether it escapes intercurrent infections and upsets during the first year of life.

Hess [10] in his treatise on prematurity has compiled data from many sources which emphasize the relation between the maturity and the weight and height of these infants. In general, the greater the prematurity the more marked is the deviation of the height and weight from the normal. The measurements correspond roughly with those of normal fetuses of the same maturity.

CONCLUSIONS

It is possible that the foregoing measurements, together with the available signs of prematurity, may be of diagnostic value in uncertain cases of prematurity. Whether such measurements are characteristic of prematurity alone it is impossible to say until measurements of other conditions, such as congenital weakness in new-born infants, etc., are obtained for comparison. Up to date, I have found no condition which invalidates the presumption that these deviations from the normal are characteristic of prematurity.

10. Hess, J. H.: Premature and Congenitally Diseased Infants, Philadelphia, Lea & Febiger, 1922.

LOGARITHMIC CHARTS IN PEDIATRICS*

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NIAGARA FALLS, N. Y.

For some considerable time I, like many other pediatricians, have been dissatisfied with the charts in general use at the present time, because they represent the absolute changes only of the conditions which we intend to record graphically, but fail to represent these in their relative, or better percentages of values.

Let us take, for instance, the growth charts for normal and healthy infants taken from the chapter on growth by Robertson,[1] and we shall see that one group of male infants, whose weight at birth was 5 pounds 6 ounces (2,438 gm.), will weigh 14 pounds 2 ounces (6,407 gm.) at the completion of the first year; a second group whose weight at birth was 6 pounds 4 ounces (2,841 gm.) will weigh 16 pounds 6 ounces (7,433 gm.) at the completion of the first year; a third group which he calls the standard average, whose weight at birth was 7 pounds 4 ounces (3,289 gm.), will weight 19 pounds 2 ounces (9,675 gm.) at the completion of the first year; a fourth group whose weight at birth was 8 pounds 4 ounces (3,742 gm.) will weigh 21 pounds 10 ounces (9,809 gm.) at the completion of the first year; and finally a fifth group, whose weight at birth was 9 pounds 8 ounces (4,309 gm.), will weigh 24 pounds 12 ounces (11,226 gm.) at the completion of the first year.

If we trace these weights on the ordinary charts now in common use, the same as Robertson's (Chart 1), then we shall get the impression that the development of a male infant is the more rapid and, therefore, the more favorable the greater its birth weight.

Practically the same is true for female infants (Chart 2).

Let us, however, trace these same weights on logarithmic charts, of which extensive use is made at the present time in the industries, where production, cost and other important items are to be expressed in percentages, and we shall at once observe that the rate of growth of these groups of infants runs parallel and that, therefore, their development is identical, no matter what their weight at birth, provided naturally that it has not been influenced by intercurrent diseases; and we also see that it is immaterial at which point of the chart we register our birth weights (Charts 3, 4 and 5).

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* Received for publication, Feb. 27, 1924.

1. Robertson, T. B.: Abt's Pediatrics, Philadelphia, W. B. Saunders Company, 1923.

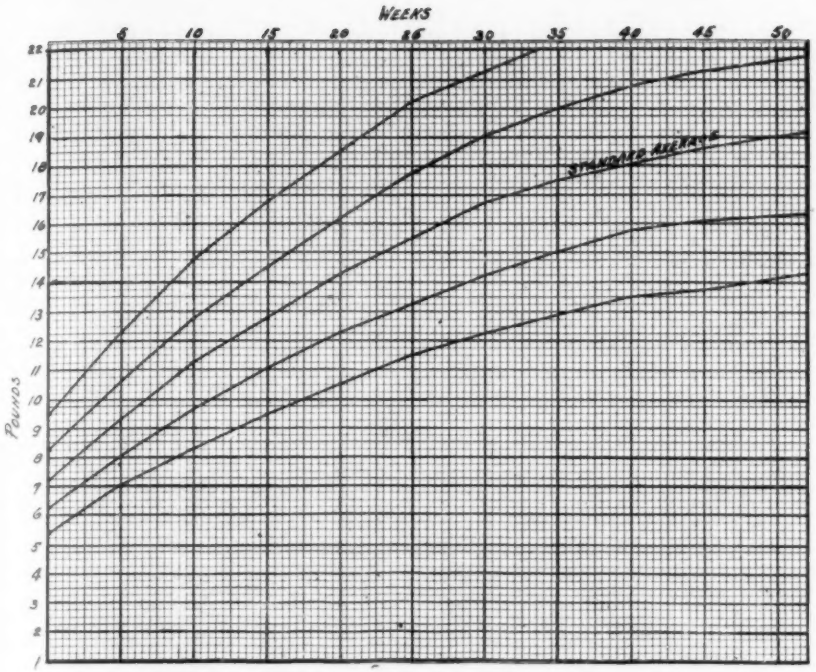

Chart 1.—Growth chart for male infants (Robertson).

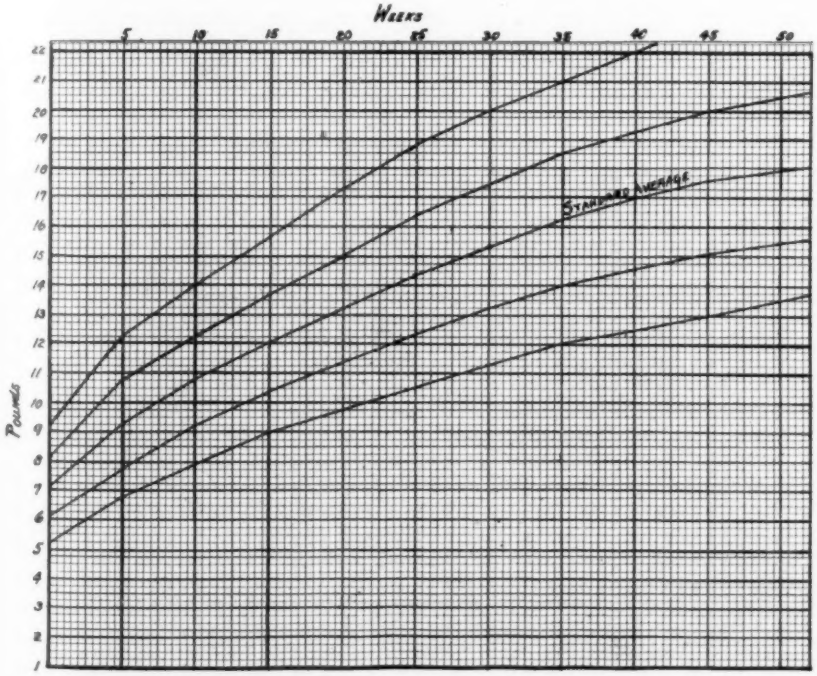

Chart 2.—Growth chart for female infants (Robertson).

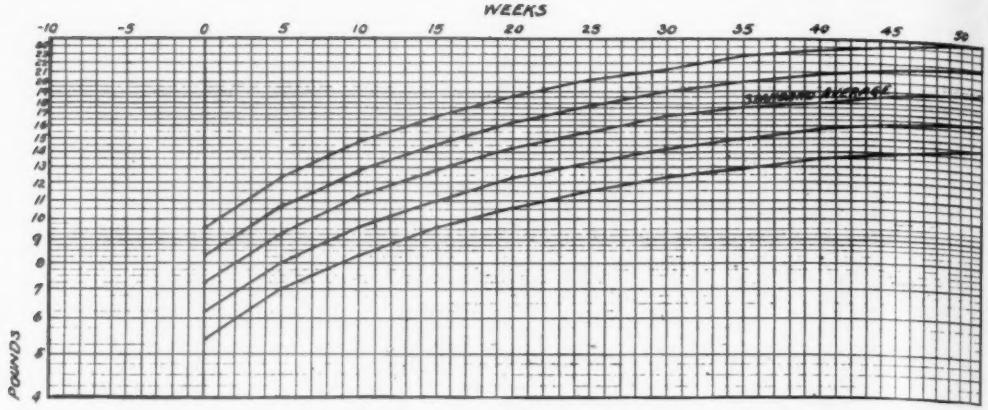

Chart 3.—Logarithmic chart, showing growth rate for male infants.

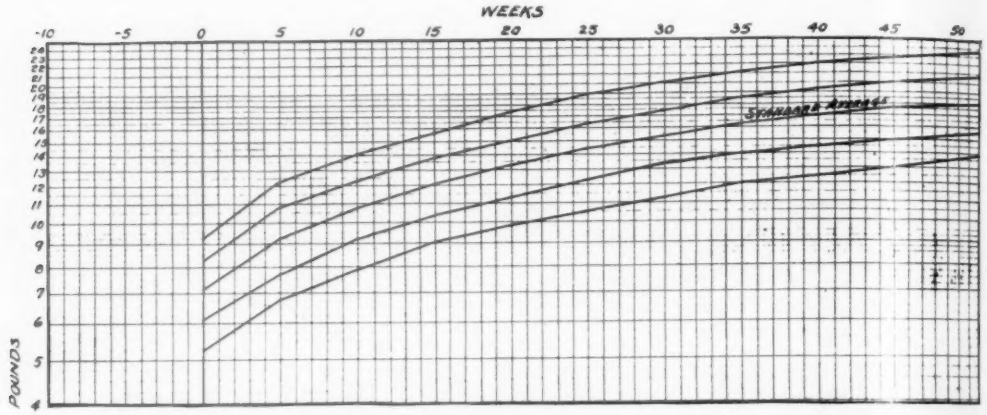

Chart 4.—Logarithmic chart, showing growth rate for female infants.

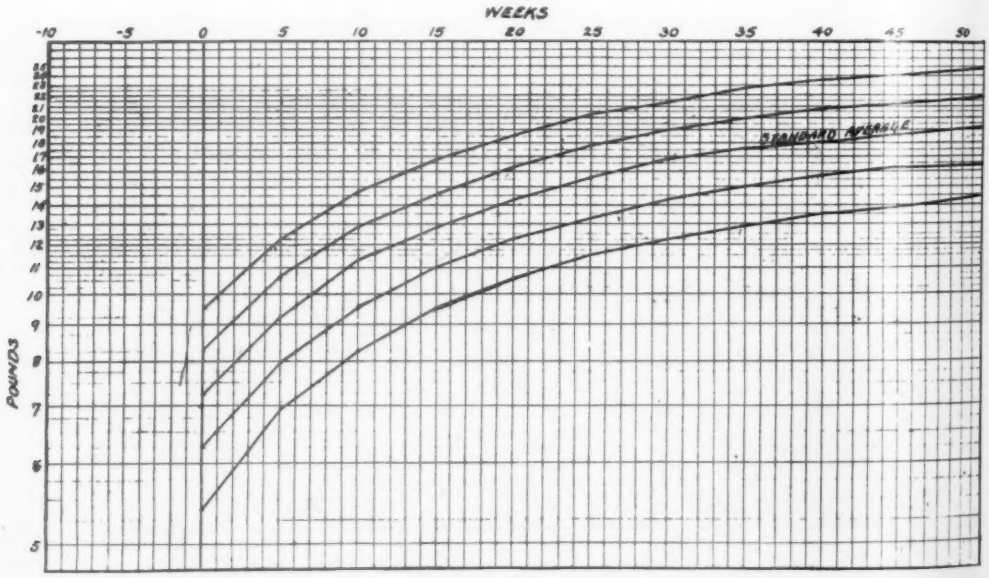

Chart 5.—Same as Chart 3.

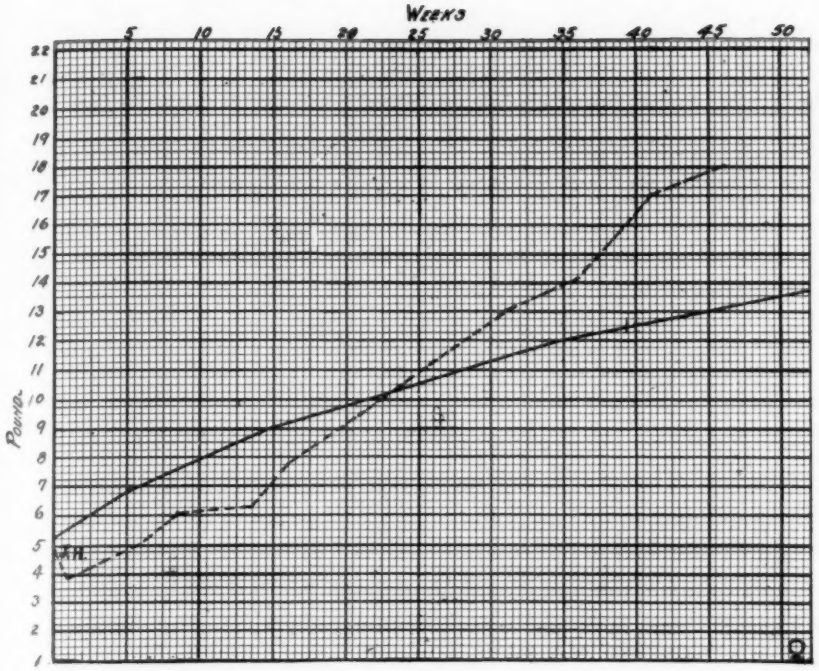

Chart 6.—Growth rate of female premature infant (broken line) compared with normal infant (straight line).

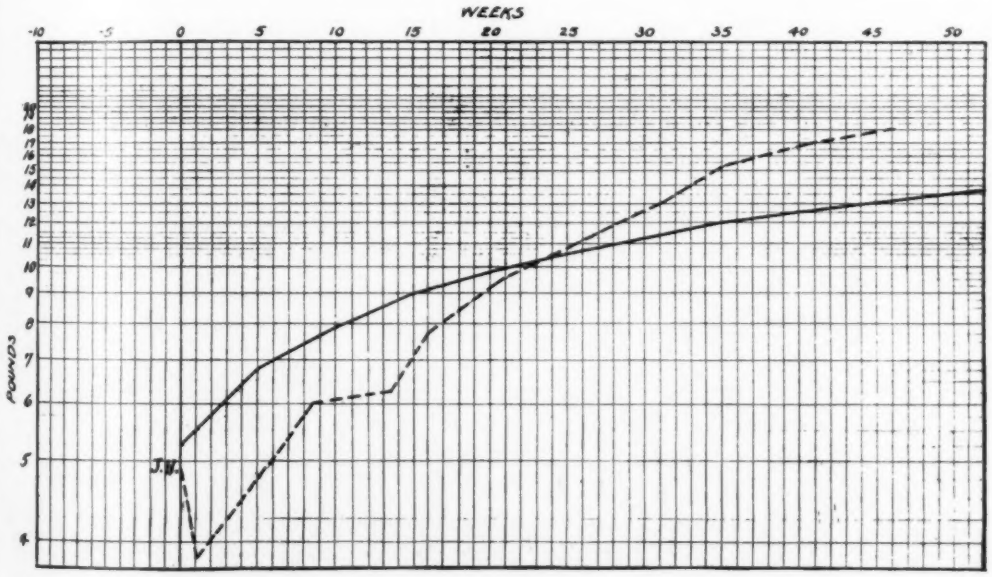

Chart 7.—Growth rate of same infants on logarithmic chart.

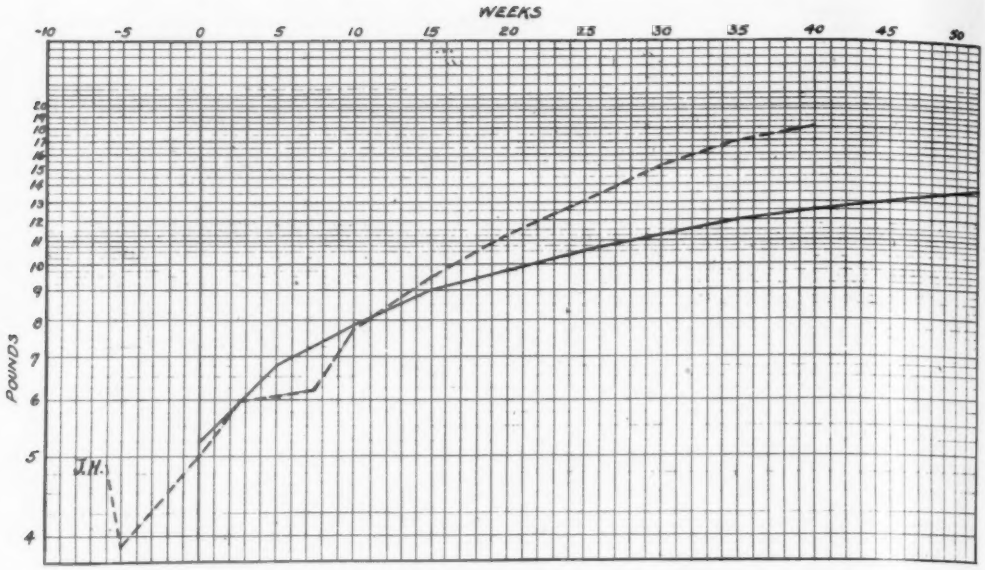

Chart 8.—More rapid development of prematurely born infant (broken line) than of normal infant (straight line).

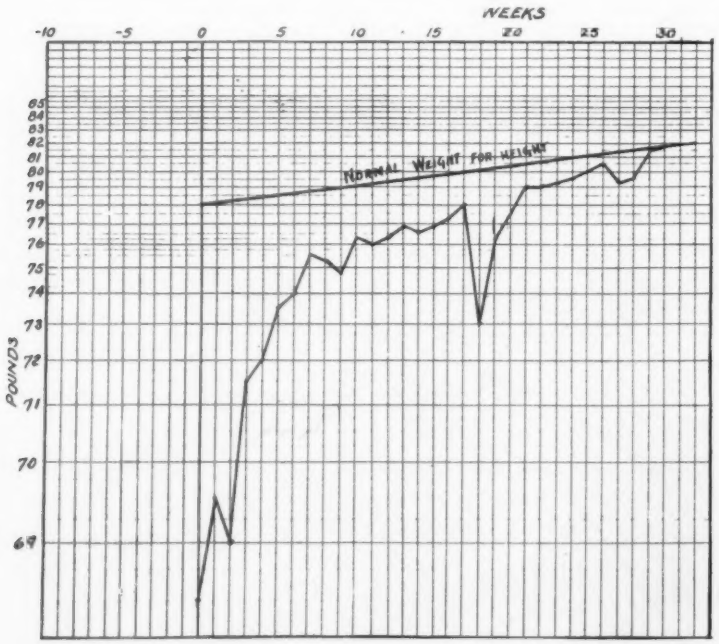

Chart 9.—Logarithmic chart of so-called "pretubercular" child.

The importance of recording the percentages of growth will be still more evident when we have to deal with infants whose weight at birth was abnormally low, due either to congenital debility or to premature birth; take, for instance, the weight chart of one of my little patients, J. H., who, though perfectly healthy, was born six weeks prematurely, and trace her weight on one of the charts now commonly in use together with that of a normal infant whose weight at birth was 5 pounds 4 ounces (2,381 gm.) (Chart 6) and compare these weights with a logarithmic chart (Chart 7) and we shall see that, according to the logarithmic chart the development of the premature infant (J. H.) was more favorable than that of the normal infant up to the thirty-fifth week of intra-uterine life, but that afterward the rate of development was about the same as that of the normal infant.

In drawing these charts of the prematurely born infants we do not, however, take into account the handicap under which the premature infant is laboring when we compare its rate of development with that of a normal infant born at term; in the former the physiologic birthday will fall on the day it should have been born if carried to full term, in the case of my patient (J. H.) six weeks after actual birth, while in the normal infant born at term the actual and the physiologic birthday are indentical.

I have, for this reason, placed the physiologic birthday on my logarithmic charts at zero, and I have left space to record the actual birth of prematurely born infants to the left of this zero, up to ten weeks, that is, up to where we may expect to have a viable fetus, so that in the case of my patient the actual birthday would be recorded at minus 6 weeks; we shall then see that, with the exception of a set back between the eighth and fourteenth week of her life, or between the second and eighth week after her physiologic birthday, the development of this prematurely born infant was much more rapid, when expressed in percentages, than that of her normal control (Chart 8).

In order to show that these charts are applicable to older children as well, I have recorded the chart of a so-called "pretubercular" child on one of my logarithmic charts (Chart 9); this chart was taken from Emerson's book.[2]

Further uses for these logarithmic charts can be developed with their employment. They may be readily prepared in any size desired with the aid of an ordinary slide rule; the originals for the charts here copied are size 6¾ by 10⅛ inches (15.6 by 25.4 cm.), as I have found this size most advantageous after extensive experiments. They can be copied at a nominal expense by photostat.

2. Emerson, W. R. P.: Nutrition and Growth in Children, pp. 126 and 127, New York, D. Appleton & Company, 1922.

REPORT OF A CASE OF GANGRENE OF LEGS FOLLOWING BRONCHOPNEUMONIA *

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This case is of special interest for three reasons; first, because of the infrequency of gangrene of the extremities following pneumonia; second, because the gangrene occurred in both legs at about the same time and to the same extent; third, because the emboli causing the gangrene were located in about the same place in the popliteal arteries.

After reviewing the literature on this subject, several causes are given for gangrene in children, namely, burns,[1] local actions of chemicals, diseases of the nervous system (Raynaud's disease), constitutional diseases (hemophilia, purpura, and diabetes), thrombosis and embolism, and gangrene following measles, scarlet fever, diphtheria, varicella, erysipelas and typhoid fever. I was unable to find a single case of bilateral gangrene following pneumonia.

As to the frequency of gangrene, Frenkel [2] after reviewing the literature found fifty cases on record following measles, diphtheria, syphilis, and other infectious diseases. Two of the cases followed pulmonary tuberculosis and only one case was bilateral.

Bull,[3] in 6,140 necropsies, found six cases of gangrene in legs caused by emboli in the main arteries; bilateral in two cases.

Kiely [4] reported a case of gangrene of the fingers of both hands caused by sclerosis of brachial arteries.

Michael [5] reported a case of gangrene of the fingers of both hands caused by obliterative arteries.

Thorp [6] reported a case of gangrene of the toes of the left foot following measles and bronchopneumonia. In this case the gangrene extended upward to the middle third of left leg, and also occurred in the right great toe before death.

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* Received for publication, March 8, 1924.

* From the department of pediatrics, University of Georgia Medical Department.

1. Cutler, G. D.: Gangrene of Toes in an Infant, Due to Scald, Am. J. Dis. Child. **21**:312 (March) 1921.

2. Frenkel, H.: Original Article: Spontaneous Gangrene of Feet in Two Children, Ztschr. f. Kinderh. **29**:213 (June 3) 1921; abstr., J. A. M. A. **77**:500 (Aug.) 1921.

3. Bull, P.: Emboli and Embolic Gangrene, Acta Chir. Scand. **54**:315 (Jan. 17) 1922; abstr., J. A. M. A. **78**:855 (March 18) 1922.

4. Kiely, C. E.: J. Lab. & Clin. Med. **3**:352 (March) 1918.

5. Michael, May: Case of Purpura with Symmetrical Gangrene of the Fingers, Am. J. Dis. Child. **20**:124 (Aug.) 1920.

6. Thorp, E.: Lancet **2**:754 (Oct. 8) 1921.

Gordon [7] reported a case of gangrene of both feet following diphtheria.

Munk [8] reported two cases of gangrene in children. The first case had gangrene of the fingers of the left hand following measles and bronchopneumonia. The toxic infectious arteritis was thought to be the cause of gangrene. The second case was gangrene of the foot following the grip.

REPORT OF CASE

History.—N. R., a negro boy, aged 6 years, entered the University Hospital on Aug. 2, 1922. The chief complaint at this time was pain in the epigastrium, disturbance in speech and tachycardia.

The patient had been sick for the last month. The trouble began with a sore throat after swimming. The cervical glands on the right side of the neck soon became swollen, the voice became nasal and there was also some swelling of the face. The child had been feverish every day.

Family History.—The father, mother and one brother were living and well. The mother had had four miscarriages before the birth of this child. There was no tuberculosis, nephritis or insanity in family.

Past Medical History.—The labor had been normal, the infant was breast fed for nine months, and had always been a healthy child. He had diphtheria at 3 years of age, epistaxis off and on all his life, and peeling of the skin on the palms of the hands and soles of the feet soon after birth. There was no history of snuffles.

Physical Examination.—The patient was a negro boy, emaciated and restless, who complained of shortness of breath. The temperature was 101; the pulse 130, and respiration 36.

The head was of normal shape; eyes, ears and nose normal; the throat not congested; the tonsils were enlarged; the voice had a nasal sound; all the neck veins were distended; the postcervical lymph glands were enlarged; dentition was fair.

The thorax showed rapid respiration with an expiratory grunting sound; the respiratory movements were shallow and about equal on both sides. There was some dulness over both bases posteriorly; a few fine crackling râles were heard over the left base posteriorly and the breath sounds were slightly tubular over the left base posteriorly. Rhonchus was present over the entire chest in front.

The heart was enlarged to the right and left, the pulmonic second sound was accentuated; there was a booming first sound, no thrill was present, a systolic murmur was heard over the entire pericardium and also heard in the left axilla. The point of maximum intensity was at the pulmonic area.

The abdomen was distended, the liver enlarged, its edge palpable six finger breadths below the costal margin in the nipple line; the spleen and kidneys were not palpable.

The extremities showed no edema of the legs, but a slight stiffness of the right ankle.

The epitrochlea and postcervical glands were enlarged.

Roentgenogram of the chest showed a greatly enlarged heart, with no evidence of fluid in either pleural cavity. Both lungs were infiltrated.

Laboratory Findings.—Blood: the white blood count was 22,000, red count, 2,700,000, hemoglobin 50 per cent., malaria smear negative, differential count showed 70 per cent. polymorphonuclears, large lymphocytes 4 per cent., small

7. Gordon, U. B., and Newman, B.: Report of a Case of Gangrene of the Feet Following Diphtheria, Am. J. Dis. Child. **23**:142 (Feb.) 1922.

8. Munk: Arch. Med. des Enf. **1**:434, 1898.

lymphocytes 26 per cent., blood platelets, 296,000, coagulation time, five minutes.

The Wassermann test was negative; the sputum negative; the throat culture negative for diphtheria bacillus.

The urine showed albumin three plus, sugar negative, microscopic examination showed a few hyaline and granular casts and pus cells.

Diagnosis at this time was as follows: Bronchopneumonia, cardiac dilatation and hepatic and renal congestion.

On August 9, the thirteenth day in hospital, the patient had a temperature of 100, pulse 120, respiration 26. Examination of the legs showed definite gangrene, dry type, involving all the toes on the left foot. He also had gangrene beginning in all the toes on the right foot. The pulse in the left dorsalis pedis artery was slightly diminished; a normal pulse was obtained in the right dorsalis pedis artery. The patient at this time complained of tenderness and pain in both legs.

On August 15, two days later, the patient had a temperature of 100, pulse 118, respiration 28. White blood count 18,000; urine negative.

The lungs at this time were clear. The heart was normal in size and no murmurs were audible.

The abdomen was negative. The patient showed marked emaciation, and kept his legs flexed on the thighs and the thighs flexed on his body. He complained of severe pain in both legs and knees when legs were extended. Gangrene had extended to the middle third of both legs and a line of demarkation was present on both sides. No femoral or popliteal pulse could be palpated. A roentgenogram of both legs did not show any arterial occlusion. Amputation of both legs above the knees was advised by the surgical staff at this time.

On September 26, both legs were amputated above the knees by Dr. Chaney. After operation, the patient gained weight and was discharged from the hospital as "well," on Nov. 2, 1922.

The pathologic report was as follows: Embolic blocking by portion of thrombus of both popliteal arteries. Gangrene of both feet and lower two-thirds of legs. The gangrene of the feet was the dry type; that of the legs was the wet type. The embolus in the right popliteal artery was lodged at the origin of the anterior tibial artery; that in the left popliteal artery was a little higher up behind the knee.

POSTENCEPHALITIC BEHAVIOR DISTURBANCE WITHOUT PHYSICAL SIGNS*

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AND
MANDEL SHERMAN, M.D.
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It has been generally recognized that postencephalitic mental disturbances in children are always preceded by some physical signs. Behavior disturbances, recognized as due to the encephalitis, because of an acute onset following a typical epidemic encephalitis history, are nearly always accompanied by some neurologic findings in the form of pupillary changes, facial paresis, muscle stiffness, etc. These behavior disturbances have been shown to be typical of an organic brain disease quite unlike the usual behavior disturbances in children. It has been demonstrated [1] that there is little or no correlation between the severity of the mental disturbance and the physical symptoms; children with slight mental disturbance may have extreme physical findings and vice versa.

Originally the behavior of a child was pronounced postencephalitic when the following three conditions were fulfilled: (1) a sudden change in personality, (2) physical evidence of postencephalitis and (3) a history of acute epidemic encephalitis. Later the third factor was not considered essential, since typical postencephalitic syndromes were frequently found without any history of an acute illness.

Children with postencephalitic behavior disturbances, however, present so typical a picture to the psychiatrist that he is often tempted to make the diagnosis even in the absence of physical findings. Since our last publication we have had under observation two children [2] presenting typical "postencephalitic behavior disturbances," in whom the physical signs appeared long afterward as typical parkinsonian syndromes; in one case, six months, and in the second case, two years after the original examination. The problem was first brought to our attention in the cases presented.

REPORT OF CASES

CASE 1.—John, a boy, aged 11 years, was referred by the Juvenile Court in January, 1923, because of "disobedience, lying, fighting, stealing and cruelty to children and animals." He was said to be "entirely uncontrollable, both at home

* Received for publication, April 2, 1924.

1. Sherman, M., and Beverly, B. I.: Deterioration in Children Showing Behavior Difficulties Following Epidemic Encephalitis, Arch. Neurol. & Psychiat. **10**:329-340 (Sept.) 1923.

2. Seen at the Institute for Juvenile Research, State of Illinois, Department of Public Welfare.

and in school." This behavior began in November, 1922, about three years after an illness, during which "he slept a considerable part of the time over a period of four or five weeks." A more adequate medical history could not be obtained. The behavior disturbance dated from the illness, but until November, 1922, only an increased irritability was noted.

Examination revealed a sluggish, apathetic appearing, but well developed and nourished boy who did not appear ill. He admitted his delinquencies readily, but could give no reason for his behavior. He had little insight into his difficulties, and appeared to have little control over his conduct. He was very shallow emotionally, and appeared entirely indifferent to the examination or his difficulties. By the Stanford-Binet intelligence tests he was found to have a mental age of 8 years and 10 months; intelligence quotient, 78. He was classified in the high borderline group. The neurologic examination was entirely negative.

In this patient, a sudden change in personality developed about three years after an illness, presumably epidemic encephalitis. The behavior and the mental symptoms being similar to that observed in a large series of postencephalitic children, a diagnosis of "probable postencephalitic behavior disturbance" was made.

Six months later the boy returned for a second examination. The behavior was unchanged. The psychologic examination revealed no evidence of a mental deterioration. The psychiatric findings were similar to those noted at the time of the first examination. The neurologic findings, however, were: slow gait; left arm immobile and slightly flexed when walking; a masked expression; unequal, irregular, sluggish pupils; tremor of the fingers. None of these findings was present at the time of the first examination. There was no history of an intervening illness.

CASE 2.—James, a boy, aged 15 years, was first examined in June, 1921. He was referred by the Board of Education with the following history. Until September, 1920, he was never considered a school problem; since that time he was reported to the principal of the school several times for indecent exposures. "He was incorrigible, and a great nuisance to the teachers." By the Stanford-Binet intelligence examination he was found to have a mental age of 11 years and 6 months; intelligence quotient, 86. The psychiatric examination revealed chiefly emotional shallowness and instability. He did not deny the behavior in school, but could give no reason for his conduct. His behavior appeared very childish for a boy with his mental age. The medical history, with the exception of frequent colds, was entirely negative. The pupils were slightly irregular, unequal and sluggish. The physical examination was otherwise negative. Because of the pupillary findings, a tentative diagnosis of syphilis of the central nervous system was made. The blood Wassermann was negative. A spinal puncture was advised but not performed.

Two years later the patient was returned by the Juvenile Court for a second examination. There was little or no change in his behavior. However, he now appeared very dull; the expression was mask-like and for this reason he was diagnosed as feebleminded by a local physician. By mental tests, however, his mental age was found to be 15 years, 4 months, intelligence quotient, 99, indicating adequate intelligence. There was no evidence of deterioration in intelligence. The psychiatric examination revealed a dull, emotionally shallow boy having little insight into his situation and unable to give any explanation for his delinquencies. The physical examination now gave clear-cut evidence of a postencephalitic syndrome. There was a slow, stiff gait; a masked expression; markedly unequal, irregular, sluggish pupils; gross tremor of the fingers; facial paresis, and a cogwheel resistance in the extremities. These findings were not present at the time of the first examination, excepting the pupillary changes which were now much more evident. The blood Wassermann was negative.

In a well developed case of behavior disturbance following epidemic encephalitis, there are both gross personality changes and distinct physical evidence of an organic brain disease. The explanation for marked personality changes preceding the physical signs is highly theoretical. From Bassoe's [3] analogy of postencephalitic syndromes with tertiary syphilis involving the central nervous system we may assume that the mental symptoms are the person's reaction to the toxic effects of the disease virus before the onset of degenerative changes. Bassoe states (in comparing syphilis to encephalitis) that "The most interesting point connecting these two diseases is the apparent existence in encephalitis as well of a 'para' stage, i. e. of progressive cerebral diseases coming on after the acute illness, and separated from it by an apparently normal interval. This is particularly true of postencephalitic parkinsonian syndrome. . . . The view is gaining ground that the virus, like that of syphilis, may remain dormant for years, and then give rise to a more chronic and degenerative process than the one encountered in the typical disease." Nonne [4] is evidently of the same opinion. He "attributes the various symptoms (postencephalitic) to the presence of a special toxin that affects the nervous centers very much like the metasyphilitic toxin." Other explanations would be that the mental symptoms result from degenerative changes that produce no immediate neurologic signs; that they are due to repeated mild infections productive of no clinical findings, or that they are due to a very chronic stage of the original acute infection.

COMMENT

A sufficient number of postencephalitic behavior disturbance cases have been studied to enable us to recognize a fairly constant symptomatology. A history of influenza or an undiagnosed condition with diplopia, a permanently developed strabismus, or a protracted period of somnolence is frequently obtained and should immediately arouse suspicion. The mental and personality changes may date from the illness or develop after a period of time even to several years. Personality changes manifested by a change in disposition, inability to sleep at night, violent outbursts of temper, irresponsibility, incorrigibility in school, hindrance in school work, cruelty to animals, lying, etc., are the most frequent complaints. The delinquencies are nearly always committed alone and are influenced but slightly by changes in environment. Deterioration of intelligence, poor memory and poor attention are often reported by teachers and parents. The psychologic examination,

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3. Bassoe, Peter: The Diagnosis of Epidemic Encephalitis, J. A. M. A. **79**:2223 (Dec. 30) 1922.

4. Nonne: Extracts, Congress of the German Society for Internal Medicine, Vienna, April, 1923, abstr. J. A. M. A. **80**:1629 (June 2) 1923.

however, gives no evidence of deterioration in intelligence. The attention and other mental capacities are normal, except for changes due to the emotional disturbance. The general impression after observing their behavior in the examining room is that the intelligence is low. The childish mannerisms, impulsive nature of the delinquencies, poor insight into their mental difficulty, the appearance of deterioration, are in marked contrast with the high rating obtained by intelligence tests. This, we think, is the most outstanding characteristic. Moreover, all these findings are referred to a definite time of onset. In a large percentage of children there is hyperactivity, irritability, alertness and marked emotional instability, the emotional tone varying from extreme anger to a cordial attitude within a few seconds. In a much smaller percentage (two of twenty cases in our first series) there was a marked indifference, lack of initiative, and sluggish bodily activity. Often there is paroxysmal polypnea with the emotional outbreaks. In one case (in a series of forty cases) there was mental difficulty manifested by extremely slow comprehension. In another case marked weakness of the lower extremities was the initial complaint.

CONCLUSION

Even in the absence of definite neurologic findings or a history of an acute disease, or both, when a child shows a sudden change of personality and symptoms of impulsive delinquencies and childish mannerisms, the appearance of deterioration in contrast with a high mental test rating, explosive reaction, with general hyperkinesis and irritability, or marked indifference with sluggish physical reactions, the possibility of an organic brain disease, especially postencephalitis, should be considered.

A COMPARISON OF THE METABOLISM OF SOME MINERAL CONSTITUENTS OF COW'S MILK AND OF BREAST MILK IN THE SAME INFANT*

II. CHLORID METABOLISM

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Chlorid metabolism experiments were conducted at the same time as those on calcium, previously reported,[1] and on the same subjects. We were unable to find that any work has been done with breast milk and cow's milk, using the same infant as subject. The literature on chlorid metabolism, therefore, deals with different subjects on a breast milk diet and others on a diet with cow's milk. Blauberg[2] compared the relative utilization of chlorid in diluted and undiluted cow's milk with that of breast milk. He used one subject for each kind of milk, and found the highest utilization in breast milk, 93.1 per cent. With undiluted cow's milk the figure was 81.8 per cent. and with modified cow's milk 59.4 per cent. He found that chlorids were utilized more completely than any other of the minerals.

Hoobler[3] calls attention to the fact that sodium chlorid has the most varied and important functions to perform, such as the maintenance of osmotic pressure and the regulation of the retention and excretion of water. The utilization is usually above 90 per cent. of the intake in both kinds of milk, according to this investigator, while the retention of sodium chlorid is only sufficient under normal conditions to cause a balance to be maintained in the body fluids. Chlorid excretion takes place almost entirely through the urine, the amount usually running about 2 gm. daily, whereas only 0.1 gm. daily is excreted through the

* Received for publication, March 8, 1924.
* From the Sarah Morris Hospital for Children, the Gusta Morris Rothschild Fund of the Michael Reese Hospital and the Nelson Morris Memorial Institute for Medical Research.

1. Wang, C. C.; Witt, D. B., and Felsher, A. R.: A Comparison of the Metabolism of Some Mineral Constituents of Cow's Milk and of Breast Milk in the Same Infant. I. Calcium Metabolism, Am. J. Dis. Child. **27:** (April) 1924.

2. Blauberg, M.: Experimentelle Beiträge zur Frage den Mineralstoffwechsel beim künstlich ernährten Säugling, Ztschr. f. Biol. **40:**1, 1900; Ueber den Mineralstoffwechsel beim natürlich ernährten Saugling, Ztschr. f. Biol. **40:**36, 1900.

3. Hoobler, B. R.: The Rôle of Mineral Salts in the Metabolism of Infants, Am. J. Dis. Child. **2:**107 (Aug.) 1921.

feces. Baumgarten [4] reports that the sodium chlorid excretion is independent of the intake, but he worked with pathologic subjects. Freund [5] on the other hand, finds that sodium chlorid excretion cannot be considered apart from the intake.

Description of Experiment.—The excretions were prepared as described in the previous paper.[1] For the determinations of sodium chlorid in feces, about 0.5 gm. of the dried and powdered feces was used. This was weighed into a silicon crucible, 2 c.c. of 5 per cent. sodium carbonate was added, and the material dried and ignited at a dull red temperature for one hour in an electric furnace. The ash was taken up in hot water and the chlorid precipitated with standard silver nitrate. The excess of silver nitrate was titrated against standard ammonium thiocyanate, using ferric ammonium sulphate as indicator. This, with some modifications, is the official method of the agricultural chemists for chlorids in beef extract.[6]

For chlorids in urine the method outlined by Harvey [7] was used without modification. The chlorids were precipitated with standard silver nitrate, and the excess of silver nitrate titrated against standard ammonium thiocyanate, using ferric ammonium sulphate as indicator.

The method developed by Denis and Sisson [8] was used with some modification for the determination of the chlorin intake in milk. To 10 c.c. of the milk 20 c.c. of 1.2 per cent. picric acid and 20 c.c. of standard silver nitrate were added, and the whole was centrifuged for five minutes. To 10 c.c. of the clear supernatant liquid was added 2 c.c. of ferric ammonium sulphate as indicator, and the excess of silver nitrate was titrated against standard ammonium thiocyanate. The experiments were made in triplicate and the results averaged.

COMMENT

It will be seen from Table 1 that although there is not always a close check in the chlorid content of the feces, the daily variation in chlorid utilization is not very great, since most of the chlorid is eliminated through the urine, as Hoobler [3] has also found. For convenience in comparison with the work of others the averages of the three-day periods are used as given in Table 2.

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4. Baumgarten, O.: Ueber "organisch" gebundenes Chlor im Harn, Zeit. f. exp. Path. u. Therap. **5**:540, 1908-1909.

5. Freund, W.: Chlor und Stickstoff im Säuglings-organismus, Jahrb. f. Kinderh. **48**:137, 1898.

6. Bureau of Chemistry Bulletin 107 (Revised), U. S. Department of Agriculture, 1912, p. 114.

7. Harvey, S. C.: The Quantitative Determination of the Chlorids in the Urine, Arch. Int. Med. **6**:12 (July) 1910.

8. Denis, W., and Sisson, W. R.: A Study of the Chlorin Content of Milk and Blood After the Ingestion of Sodium Chlorid, J. Biol. Chem. **46**:483 (May) 1921.

TABLE 1.—*Daily Chlorid Metabolism in Healthy Infants Fed on Breast Milk and Cow's Milk*

TABLE 1.—*Daily Chlorid Metabolism in Healthy Infants Fed on Breast Milk and Cow's Milk*

Name	Kind of Milk	Date	NaCl Intake: Per 24 Hours, Gm.	NaCl Intake: Per Kg., Gm.	NaCl Output: Feces per 24 Hours, Gm.	NaCl Output: Urine per 24 Hours, Gm.	NaCl Output: Total per 24 Hours, Gm.	NaCl Output: Total per Kg. per 24 Hrs., Gm.	NaCl Utilization: Per 24 Hours, Gm.	NaCl Utilization: Per Kg. per 24 Hours, Gm.	NaCl Utilization: Per Cent. of Intake	NaCl Retention: Per 24 Hours, Gm.	NaCl Retention: Per Kg. per 24 Hours, Gm.	NaCl Retention: Per Cent. of Intake
W. O.	Breast	11/26/21	0.4036	0.0977	0.0806	0.2566	0.3372	0.0816	0.3230	0.0782	80.02	0.0664	0.0161	16.45
		11/27/21	0.4036	0.0977	0.1416	0.1878	0.3294	0.0798	0.2620	0.0634	64.91	0.0742	0.0180	18.38
		11/28/21	0.4109	0.0995	0.1156	0.1800	0.2956	0.0716	0.2953	0.0715	71.86	0.1153	0.0279	28.06
	Cow's	12/ 7/21	1.3010	.....	0.1868	1.1251	1.3119	0.2988	1.1142	0.2537	85.58	—0.0109	—0.0025	.....
		12/ 8/21	1.3010	0.2964	0.0974	0.7218	0.8192	0.1866	1.2036	0.2743	92.52	0.4818	0.1097	37.02
		12/ 9/21	1.3010	.....	0.1820	0.8166	0.9986	0.2275	1.1190	0.2548	85.96	0.3024	0.0689	23.23
	Breast	12/16/21	0.5456	.....	0.0216	0.2282	0.2498	0.0560	0.5240	0.1175	96.03	0.2958	0.0663	53.73
		12/17/21	0.5456	0.1224	0.0713	0.2782	0.3495	0.0784	0.4743	0.1064	86.94	0.1961	0.0440	35.94
		12/18/21	0.5456	.....	0.0626	0.2895	0.3721	0.0834	0.4630	0.1039	84.86	0.1736	0.0389	31.79
E. M.	Cow's	11/26/21	1.5420	0.2075	0.3388	2.0168	2.3556	0.3169	1.2032	0.1619	78.01	—0.8136	—0.1095	.....
		11/27/21	1.6400	0.2208	0.2937	0.9249	1.2186	0.1640	1.3463	0.1811	82.06	0.4214	0.0567	25.70
		11/28/21	1.6630	0.2238	0.2894	0.9110	1.2004	0.1616	1.3736	0.1849	82.66	0.4626	0.0623	27.83
	Breast	12/ 3/21	0.5334	0.0728	0.1890	0.3569	0.5459	0.0745	0.3444	0.0470	64.56	—0.0125	—0.0017	.....
		12/ 4/21	0.5334	.....	0.0370	0.3586	0.3956	0.0540	0.4964	0.0677	93.08	0.1378	0.0188	25.84
T. O.	Breast	12/13/21	0.3816	.....	0.0015	0.3577	0.3592	0.1009	0.3795	0.1066	99.63	0.0218	0.0061	5.72
		12/14/21	0.3816	0.1070	0.0015	0.3577	0.3592	0.1009	0.3795	0.1066	99.63	0.0218	0.0061	5.72
		12/15/21	0.3816	.....	0.0182	0.4008	0.4190	0.1177	0.3628	0.1020	95.26	—0.0380	—0.0107	.....
	Cow's	1/14/22	1.4350	.....	0.1305	1.0420	1.1725	0.2902	1.3045	0.3207	90.30	0.2625	0.0650	18.29
		1/15/22	1.4350	0.3551	0.1305	1.0420	1.1725	0.2902	1.3045	0.3207	90.30	0.2625	0.0650	18.29
		1/16/22	1.4350	.....	0.0780	1.1834	1.2614	0.3122	1.3570	0.3359	94.58	0.1736	0.0430	12.10
B. N	Cow's	3/ 9/22	1.2323	.....	0.0099	0.8942	0.9041	0.2773	1.2224	0.3749	99.20	0.3282	0.1007	26.65
		3/10/22	1.2323	0.3779	0.0330	0.8121	0.8451	0.2593	1.1993	0.3679	97.34	0.3872	0.1188	31.43
		3/11/22	1.2323	.....	0.0137	0.7405	0.7542	0.2314	1.2186	0.3739	98.93	0.4781	0.1450	38.81
	Breast	3/30/22	0.3375	.....	0.0207	0.2197	0.2404	0.0664	0.3168	0.0875	93.88	0.0971	0.0268	28.77
		3/31/22	0.3375	0.0932	0.0207	0.2197	0.2404	0.0664	0.3168	0.0875	93.88	0.0971	0.0268	28.77
		4/ 1/22	0.3375	.....	0.0647	0.2379	0.3021	0.0834	0.2733	0.0755	81.00	0.0354	0.0098	10.49
B. B.	Cow's	2/25/22	1.5670	.....	0.0352	1.0780	1.1132	0.2914	1.5318	0.4011	97.78	0.4538	0.1188	28.96
		2/26/22	1.5670	0.4102	0.0352	1.0780	1.1132	0.2914	1.5318	0.4011	97.78	0.4538	0.1188	28.96
		2/27/22	1.5670	.....	0.0630	1.3151	1.3781	0.3607	1.5040	0.3937	95.96	0.1889	0.0495	12.06
	Breast	3/ 8/22	0.4477	.....	0.0088	0.2888	0.2976	0.0746	0.4389	0.1100	98.04	0.1501	0.0376	33.53
		3/ 9/22	0.4477	0.1122	0.0168	0.3440	0.3608	0.0904	0.4309	0.1080	96.25	0.0869	0.0218	19.41
		3/10/22	0.4477	.....	0.0247	0.4090	0.4337	0.1087	0.4230	0.1060	94.48	0.0140	0.0035	3.13
	Cow's	3/29/22	1.7400	.....	0.0020	1.4065	1.4085	0.3172	1.7380	0.3913	99.88	0.3315	0.0746	19.05
		3/30/22	1.7400	0.3948	0.0559	1.3972	1.4531	0.3273	1.6841	0.3793	96.80	0.2869	0.0646	16.49
		3/31/22	1.7400	.....	0.0559	1.3972	1.4531	0.3273	1.6841	0.3793	96.80	0.2869	0.0646	16.49
	Breast	4/11/22	0.4577	.....	.....	0.3631	.....	.....	.....	.....	.....	.....	.....	.....
		4/12/22	0.4577	0.0982	0.0037	0.8512	0.8549	0.1835	0.4540	0.0974	99.20	—0.3972	—0.0852	.....
		4/13/22	0.4577	.....	.....	0.3821	.....	.....	.....	.....	.....	.....	.....	.....

TABLE 2.—*Average Daily Chlorid Metabolism of Healthy Infants Fed on Breast Milk and Cow's Milk*

Name	Age, Mo.	Body Weight	Kind of Milk	Fat	NaCl Intake		NaCl Output				NaCl Utilization			NaCl Retention		
					Per 24 Hours, Gm.	Per Kg., Gm.	Feces per 24 Hours, Gm.	Urine per 24 Hours, Gm.	Total per 24 Hours, Gm.	Total per Kg. per 24 Hours, Gm.	Per 24 Hours, Gm.	Per Kg. per 24 Hours, Gm.	Per Cent. of Intake	Per 24 Hours, Gm.	Per Kg. per 24 Hours, Gm.	Per Cent. of Intake
W. O.	2.6	4.13	Breast	4.0	0.4060	0.098	0.1126	0.2081	0.3207	0.078	0.2934	0.071	72.3	0.0853	0.021	21.0
	3.0	4.39	Cow's	...	1.3010	0.296	0.1554	0.8878	1.0432	0.238	1.1456	0.261	88.1	0.2578	0.059	19.8
	3.3	4.46	Breast	4.4	0.5456	0.122	0.0585	0.2653	0.3238	0.073	0.4871	0.109	89.3	0.2218	0.050	40.7
E. M.	5.5	7.43	Cow's	2.2	1.6150	0.217	0.3073	1.2842	1.5915	0.214	1.3077	0.176	81.0	0.0704	0.010	4.4
	5.7	7.33	Breast	3.8	0.5334	0.073	0.1130	0.3577	0.4707	0.064	0.4204	0.057	78.8	0.0626	0.009	11.7
T. O.	3.2	3.56	Breast	4.0	0.3810	0.107	0.0071	0.3721	0.3791	0.107	0.3739	0.105	98.1	0.0019	0.001	0.5
	4.3	4.04	Cow's	2.2	1.4350	0.355	0.1130	1.0891	1.2021	0.298	1.3220	0.326	92.1	0.2329	0.058	16.2
B. N.	0.4	3.26	Cow's	2.1	1.2323	0.378	0.0189	0.8156	0.8345	0.256	1.2134	0.372	98.5	0.3978	0.122	32.3
	1.4	3.62	Breast	3.6	0.3375	0.093	0.0352	0.2258	0.2610	0.072	0.3023	0.084	89.6	0.0765	0.022	22.7
B. B.	1.3	3.82	Cow's	2.0	1.5670	0.410	0.0445	1.1157	1.2015	0.315	1.5238	0.598	97.2	0.3655	0.096	23 3
	1.6	3.99	Breast	4.2	0.4477	0.112	0.0168	0.3473	0.3640	0.091	0.4309	0.108	96.2	0.0837	0.021	18.7
	2 1	4.44	Cow's	2.2	1.7400	0.395	0.0379	1.4003	1.4382	0.324	1.7021	0.383	96.1	0.3018	0.068	17.3
	2.4	4.66	Breast	3.4	0.4577	0.098	.....	0.5321	.....	.....	.....	.....	....	.....	.....	....

Difference in the Quantity of Chlorid Excretion in Feces and Urine Brought About by the Change of Milk.—The chlorid intake in our cases is invariably much greater on a diet of cow's milk than of breast milk; with cow's milk it varied from 1.2323 gm. to 1.7400 gm. per twenty-four hours, with an average of 1.4817 gm. The corresponding figures for breast milk are 0.3810 gm. to 0.5456 gm. with an average of 0.4441 gm. These figures are in general higher than those of other investigators. Table 3 shows figures reported by other investigators working on breast fed infants. The range here is from 0.1140 gm. to 0.4586 gm. per twenty-four hours. Table 3 also gives figures for cow's milk from three investigators. Blauberg [2] had a lower chlorid intake than ours, Hoobler [3] higher, but Tangle's [9] figures are much like ours.

A change from one milk to the other is always accompanied by a change in the chlorid excretion both in feces and in urine. With the exception of one case (B. N.), the value for feces is always higher for cow's milk than for breast milk. There is a great variation in different individuals in the sodium chlorid content of feces; with breast milk the values varied from 0.0071 gm. to 0.1130 gm. per twenty-four hours, with an average of 0.0572 gm. With cow's milk it ran from 0.0189 gm. to 0.3073 gm. with an average of 0.1128 gm. Other investigators show a range of sodium chlorid content in feces of from 0.0045 gm. to 0.1210 gm., which is even greater than ours. The four cases of cow's milk reported in Table 3 also show a marked variation, from 0.042 gm. to 0.1170 gm. There is no consistent relation between the intake and excretion of sodium chlorid in the feces, either in our work or in the previous work reported.

An increase of sodium chlorid intake leads to a decided increase in the output of sodium chlorid in the urine. Both are invariably higher on a diet of cow's milk and every change from breast milk to cow's milk brought about an increase in urinary sodium chlorid. The breast fed infants excreted from 0.2081 gm. to 0.5321 gm. urinary sodium chlorid per twenty-four hours, with an average of 0.3298 gm. With cow's milk the figures were 0.8156 gm. to 1.4033 gm., with an average of 1.0988 gm. In every case when the milk is changed the increase or decrease of urinary sodium chlorid is threefold or more. In the case of B. B., on whom the experiment was repeated with both diets, the excretion increased with age on breast milk and on cow's milk, although the intake per kilogram in the second period was lower both times. However, comparing different infants, there is no apparent relationship between age and the amount of urinary sodium chlorid.

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9. Tangle, Franz: Der Stoff und Energieumsatz eines künstlich ernährten Säuglings, Arch. f. d. ges. Physiol. **104**:453, 1904.

TABLE 3.—*Chlorid Metabolism in Healthy Infants Fed on Breast Milk and Cow's Milk*

Author	Subject	Duration of Period, Days	Age, Mo.	Weight, Kg.	Chlorid Intake		Chlorid Output			Chlorid Utilization			Chlorid Retention		
					Per 24 Hours, Gm.	Per Kg. per 24 Hours, Gm.	Feces per 24 Hours, Gm.	Urine per 24 Hours, Gm.	Total per 24 Hours, Gm.	Per 24 Hours, Gm.	Per Kg. per 24 Hours, Gm.	Per Cent. of Intake	Per 24 Hours, Gm.	Per Kg. per 24 Hours. Gm.	Per Cent. of Intake
Breast Milk															
Freund [5]	1	4	4.5	4.94	0.280	0.056	0.011	0.208	0.219	0.169	0.034	60.21	0.061	0.012	21.75
	2	6	3.0	2.95	0.393	0.136	0.012	0.258	0.270	0.381	0.127	96.94	0.123	0.041	31.04
Tobler L., and Noll, F.: Monatschr. f. Kinderh. **9**: 210, 1910...	1	6	2.5	4.04	0.114	0.028	0.0045	0.0921	0.0960	0.1095	0.027	96.07	0.0174	0.0044	15.26
Lindberg, G.: Ztschr. f. Kinderh. **16**: 90, 1917.....	1	...	2.5	4.285	0.4189	0.098	0.0214	0.2515	0.2729	0.3976	0.093	94.90	0.1460	0.0034	34.86
				4.370	0.4208	0.096	0.0402	0.2342	0.2744	0.3806	0.087	90.45	0.1464	0.0033	34.79
				4.370	0.3053	0.070	0.0264	0.1188	0.1452	0.2789	0.063	91.35	0.1601	0.0036	52.43
				4.370	0.3215	0.073	0.0588	0.1420	0.2008	0.2627	0.060	81.71	0.1208	0.0028	37.56
Hoobler [3]	1	...	...	.....	0.3297	.....	0.0135	0.1776	0.1911	0.3162	.....	95.4	0.1386	.....	42.04
Blauberg [2]	1	6	5	.....	0.3350	.....	0.0231	0.0215	0.0456	0.3119	.....	93.1	0.2894	.....	86.4
Michel, C., and Perrett, M.: Bull. Soc. d. obst. et de gynec. **2**: 98, 1890.....	...	...	3	4.725	0.4586	0.098	0.0170	0.3284	0.3454	0.4416	0.094	96.3	0.1139	0.024	24.8
Klotz, M.: Jahrb. f. Kinderh. **70**: 1, 1909.....	...	...	2.5	3.42	0.3606	0.108	0.1210	0.0931	0.2141	0.2485	0.073	67.25	0.1555	0.046	42.05
Cow's Milk															
Hoobler [3]	...	...	9	....	2.3178	.....	0.042	2.2421	2.2841	2.2758	0.274	98.1	0.0337	0.004	1.4
Blauberg [2]	...	...	...	....	0.2850	.....	0.1170	0.3952	0.5122	0.1680	.....	59.0	−0.2272	.....	.....
Tangle [9]	...	...	3.0	4.51	1.469	0.326	0.046	1.306	1.352	1.422	0.315	96.8	0.117	0.026	7.96
			4.7	5.41	1.148	0.212	0.041	1.006	1.047	1.107	0.205	96.4	0.101	0.019	8.80

The figures of Table 3 are difficult to compare with ours. The range there tabulated, with breast milk, is from 0.0215 gm. to 0.3284 gm. per twenty-four hours. This discrepancy might well be due to the much lower intake, since more than 50 per cent. of these cases had a chlorid intake less than our minimum. In the cases where the intake of sodium chlorid approximates that of our cases the urinary sodium chlorid is also within our range, with the exception of the case reported by Blauberg,[2] where the excretion is exceptionally low and the retention unusually high. Blauberg [2] in his one case with cow's milk had a very low intake and a low excretion. Hoobler [3] reported a very high intake and a correspondingly high excretion. Tangle's [9] cases check quite well with ours.

The Effect of the Change of Milk on the Utilization and Retention of Chlorids.—Since the chlorid is mainly eliminated through the urine, the actual value of the utilization runs parallel to the urinary chlorids. As urinary chlorid depends on intake, the utilization in grams per twenty-four hours is always higher on a diet of cow's milk. With breast milk the utilization varies from 0.2934 gm. to 0.4871 gm. with an average of 0.3847 gm. In the case of cow's milk the corresponding figures are from 1.1456 gm. to 1.7021 gm. with an average of 1.3691 gm. Although the actual quantity of sodium chlorid utilized from the two milks varies so greatly, there is no marked difference in the percentage utilization. The ranges are for breast milk from 72.3 to 98.1, with an average of 87.4 per cent. as against cow's milk from 81.0 to 98.5, with an average of 92.2 per cent. Here again, the work reported in Table 3, with the exception of those cases where the intake was very low, is in fairly close agreement with our figures. Excluding two cases, their figures ran from 0.2485 gm. to 0.4416 gm. with breast milk. Their percentage utilization runs from 60.2 to 96.9.

There were three cases with breast milk and two with cow's milk in which one day during the period showed a negative retention value. However, the average of three-day periods always gave a positive value. There is a much greater variation in retention in different individuals than in utilization. With breast milk the figures range from 0.0019 gm. to 0.2218 gm. per twenty-four hours, with an average of 0.0886 gm. For cow's milk the range is from 0.0704 gm. to 0.3978 gm. with an average of 0.2710 gm. Here again, however, the average percentage values follow each other very closely, that of breast milk being 19.2 per cent. and cow's milk 18.9 per cent. So it seems that the variation in the intake is accompanied by a corresponding variation in utilization and retention in the same direction, resulting in a constant percentage. This is a fact for which it is very difficult to account.

The values for retention reported by other investigators also vary within wide limits. Their figures for breast milk run from 0.0174 gm.

to 0.2894 gm., or from 15.3 to 86.0 per cent. The four cases reported using cow's milk are again hard to compare with ours. Hoobler [3] reported a utilization of 2.2758 gm. per twenty-four hours, or 98.1 per cent. of the intake, and a retention of 0.0337 gm. or 1.4 per cent. Blauberg's [2] case gave unusually low values, utilization of 1.680 gm. per twenty-four hours, or 59.0 per cent. Tangle [9] had an average retention of 0.109 gm., or 8.38 per cent.

Relation of Age to Chlorid Utilization and Retention.—Table 4 shows that although there is no marked difference in the utilization, the actual grams retained in the body varied inversely with age; thus the youngest infant, B. N., at 0.4 months had a retention of 0.3978 gm. per twenty-four hours. The corresponding figure for E. M. at 5.5 months, was 0.0704 gm. The percentage retention varied in the same way. The actual quantity of chlorid utilized showed no consistent relation to age,

TABLE 4.—*Relation of Age to Chlorid Metabolism, Cow's Milk*

Name	Age, Months	NaCl Utilization		NaCl Retention		Blood Chlorid
		Per 24 Hours, Gm.	Per Cent. of Intake	Per 24 Hours, Gm.	Per Cent. of Intake	Per 100 C.c. of Plasma, Mg.
B. N.	0.4	1.2134	98.5	0.3978	32.3	552.9
B. B.	1.3	1.5238	97.2	0.3655	23.3	559.1
B. B.	2.1	1.7021	96.1	0.3018	17.3	540.0
W. O.	3.0	1.1456	88.1	0.2578	19.8	650.0
T. O.	4.3	1.3220	92.1	0.2329	16.2	658.0
E. M.	5.5	1.3077	81.0	0.0704	4.4	748.7

TABLE 5.—*Relation of Age to Chlorid Metabolism, Breast Milk*

Name	Age, Months	NaCl Utilization		NaCl Retention		Blood Chlorid
		Per 24 Hours, Gm.	Per Cent. of Intake	Per 24 Hours, Gm.	Per Cent. of Intake	Per 100 C.c. of Plasma, Mg.
B. N.	1.4	0.3023	89.6	0.0765	22.7	501.3
B. B.	1.6	0.4309	96.2	0.0837	18.7	601.8
W. O.	2.6	0.2984	72.3	0.0853	21.0	615.6
T. O.	3.2	0.3739	98.1	0.0019	0.5	629.3
W. O.	3.3	0.4871	89.3	0.2218	40.7	614.2
E. M.	5.7	0.4204	78.8	0.0626	11.7	804.0

but the percentage had the same tendency as that for retention. In the case of breast milk there was no relation between age and utilization and retention, as may be seen from Table 5. The tendency toward greater retention of chlorids in the younger infants might possibly be due to the more rapid growth of the younger organism and the consequent need for a larger amount of chlorid. We are unable to account for the variation in results between breast milk and cow's milk.

Relation of Blood Chlorid to the Chlorids in Breast Milk and Cow's Milk.—For blood chlorid, Rusznyák's [10] method was followed and the

10. Rusznyák, S.: Eine Methode zur Bestimmung der Chloride in kleinen Flüssigkeitsmengen, Biochem. Zeit. **114**:23, 1921.

determinations were made on the plasma. Tables 4 and 5 show no relation between the kind of milk fed and the chlorid content of the blood, but of six tests on each milk two had a higher blood chlorid with breast milk and four with cow's milk. The average figure for blood chlorid with breast milk was 627.7 mg. per hundred cubic centimeters of plasma, and that of cow's milk was 618.1 mg. per hundred cubic centimeters of plasma. The time of drawing blood was not controlled in relation to the meals, and so the varying results in several cases may be due to the time factor. Scheer [11] and Dodds and Smith [12] call attention to the fact that blood chlorids are lowered shortly after a meal, due to the gastric secretion of hydrochloric acid.

SUMMARY

1. Change of diet from breast milk to cow's milk was invariably followed by an increase in the chlorid excretion in both feces and urine, and vice versa. The increase is due, at least in part, to the increased chlorid intake in cow's milk. The average intake with cow's milk was 1.4817 gm. per twenty-four hours and with breast milk 0.4441 gm. The average excretion in feces was 0.1128 gm. and 0.0576 gm. respectively, and in urine 1.0988 gm. and 0.3298 gm.

2. The utilization of chlorids runs parallel to the urinary chlorid; with breast milk it is 0.3847 gm. per twenty-four hours and with cow's milk, 1.3691 gm. There is no marked difference in percentage utilization of chlorid intake between the two milks. For breast milk the average utilization was 87.4 per cent. as against 92.2 per cent. for cow's milk.

3. There was great variation in chlorid retention in different individuals. The average retention for breast milk was 0.0886 gm. per twenty-four hours, or 19.2 per cent., and for cow's milk 0.2710 gm. or 18.9 per cent.

4. As age increased there was a decrease in the actual amount of chlorid retained with cow's milk, but no such relation existed with breast milk; neither was there any definite relation between age and percentage retention.

5. Change of milk had no effect on the chlorid content of the blood. On a diet of breast milk the average blood chlorid was 627.7 mg. per hundred cubic centimeters of plasma, and on cow's milk it was 618.1 mg.

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11. Scheer, K.: Untersuchungen über den Chloridgehalt des Serums beim Säugling unter verschiedner Bedingungen, Jahrb. f. Kinderh. **94**:295, 1921.

12. Dodds, E. C., and Smith, K. S.: Variations in the Blood Chlorids in Relation to Meals, J. Physiol. **58**:157 (Dec.) 1923.

THE EFFECT OF FLUID ON THE TEMPERATURE AND BLOOD CONCENTRATION IN THE NEW-BORN WITH FEVER*

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In a previous paper [1] evidence was presented to show that a close relationship exists between fever in the new-born and dehydration. Whereas in afebrile new-born infants the protein concentration of blood serum is fairly constant; usually lying between 6 and 7 per cent., in the new-born with fever the serum proteins are generally above 7.5 per cent. and often over 9 per cent., indicating a marked diminution in the plasma water.

In the experiments reported in this paper the effect of fluid therapy on the temperature and blood concentration of the new-born with fever was studied. Approximately 35 c.c. of fluid per kilogram of body weight (a total of from 100 to 150 c.c.), warmed above the temperature of the baby, were given and the temperature and blood concentration determined repeatedly before and after the fluid administration. The fluid was given in various ways—orally (gavage), hypodermically and intraperitoneally. The gavage method for introducing fluid into the stomach was used so that a large amount of fluid could be given quickly. It is probable that similar results would be obtained if the fluids were given by mouth in the usual manner. Tap water, saline and glucose solution were the fluids used. The babies were kept well covered during the experiments to avoid a fall in temperature due to cooling of the body surface. Benedict and Talbot [2] have emphasized the importance of this factor in reducing the temperature of the new-born with fever. The same thermometer was used throughout each experiment.

Variations in the blood concentration were estimated from changes in the protein concentration of serum and also, in most instances, from changes in the red cell count. The technic used for determining the serum protein concentration was similar to that described in previous

* Received for publication, March 13, 1924.

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1. Bakwin, H.: Dehydration Fever in the New-Born, II., Am. J. Dis. Child. **24**:508 (Dec.) 1922.

2. Benedict, F. G., and Talbot, F.: Carnegie Institute Publications, No. 233, 1915.

papers.[3] The pipets and counting chamber used for the red cell counts were standardized by the Bureau of Standards. Dilutions were made with Hayem's solution and the mixtures shaken in a mechanical shaker.

The effect of water by mouth on the temperature and blood concentration of infants, from 2 to 15 months of age, with fever due to various infections was studied as a control group.

RESULTS

The Effect of Fluid by Mouth on the Temperature and Blood Concentration of New-Born Infants with Fever.—When warm water was given by gavage in the proportion of 30 to 40 c.c. per kilogram of

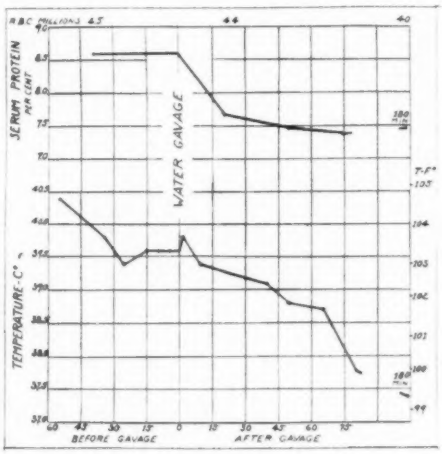

Chart 1.—Water gavage; drop in temperature and blood concentration in a new-born with fever.

Case 5 (Chart 1).—The baby, a boy, healthy at birth, developed fever on the sixth day after a weight loss of 510 gm. The mouth and tongue were dry and the skin loose and inelastic. The stools were frequent, green and watery. The serum proteins were 8.61 per cent., indicating a marked diminution in the plasma water. Following a gavage of 100 c.c. of warm water the temperature fell, reaching the normal level in two hours and remained normal during the rest of the baby's stay in the hospital. Coincident with the drop in temperature there was a fall in the serum protein concentration from 8.61 to 7.37 per cent. The red cells fell from 4,500,000 per cubic millimeter to 3,960,000.

body weight, a prompt fall in temperature and blood concentration followed with great regularity (fifteen out of sixteen cases studied). The temperature usually reached normal within an hour. The drop in serum protein concentration ordinarily amounted to about 1 per cent. of protein, representing an increase of approximately 12 per cent. in

3. Bakwin, H.: Dehydration in the New-Born, I., Am. J. Dis. Child. **24**:497 (Dec.) 1922.

the plasma water. The fall in the red cell count was more marked than would be expected from the drop in the serum protein concentration and represented, in most instances an increase of about 15 per cent. in the whole blood water. The results in four typical cases are shown in Charts 1 to 4.

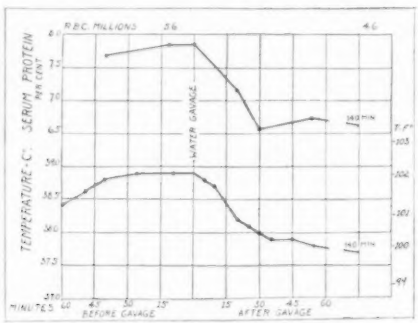

Chart 2.—Water gavage; prompt drop in temperature and blood concentration in a new-born with fever.

CASE 6 (Chart 2).—The baby, a girl, vigorous at birth, developed fever on the second day of life after a loss of 330 gm. Following a water gavage of 100 c.c. the temperature fell to normal in an hour. The serum proteins dropped from 7.84 per cent. to 6.74 and the red cells from 5,630,000 to 4,580,000 per cubic millimeter.

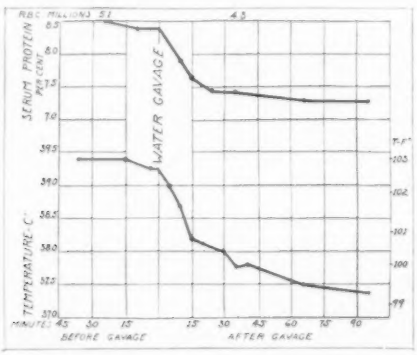

Chart 3.—Water gavage; prompt drop in temperature and blood concentration in a new-born with fever.

CASE 7 (Chart 3).—The baby, a girl, developed fever on the third day of life after a weight loss of 320 gm. The mucous membranes of the mouth were dry and red and the skin was loose and inelastic. The baby cried continuously and took the breast eagerly. The serum protein concentration was 8.55 per cent., indicating a marked decrease in the plasma water. The temperature fell to normal within an hour after a gavage of 110 c.c. of warm water. There was a simultaneous drop in the serum proteins from 8.55 to 7.31 per cent., and in the red cell count from 5,080,000 per cubic millimeter to 4,300,000. There was a secondary rise in temperature on the fifth day associated with a loss in weight and an increase in the serum protein concentration to 8.13 per cent. The temperature fell to normal within twenty-four hours after the free administration of water by mouth. There was no further rise in temperature.

The one patient in whom the water gavage failed to reduce the temperature was an infant with severe dehydration and a serum protein concentration of 10.17 per cent. Estimations of the serum proteins were not made after the fluid was given.

The results following the administration of a 5 per cent. glucose solution by gavage were similar to those obtained with water. With saline by mouth there was a tendency to diarrhea and these experiments were consequently discontinued. In one baby, due to an error, a 10 per cent. glucose solution was given instead of a 5 per cent. solution. A profuse, watery diarrhea, similar to that following the administration of a hypertonic salt solution, occurred. The stools, after the first few had been passed, contained no solid material and reduced Benedict's solution strongly. A rise in temperature accompanied the loss of fluid through the diarrhea.

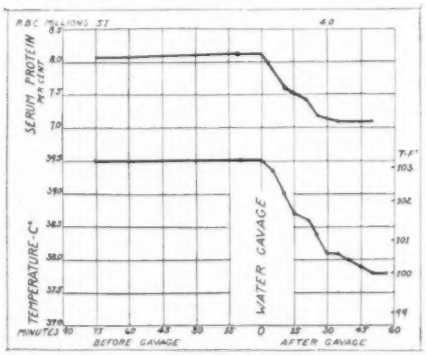

Chart 4.—Water gavage; prompt drop in temperature and blood concentration in a new-born with fever.

Case 8 (Chart 4).—The baby, a girl, developed fever on the third day of life after a weight loss of 420 gm. The skin was dry and inelastic, the mucous membranes of the mouth red and dry and the cord slightly moist. The baby cried incessantly and took the breast eagerly.

The temperature fell to normal within an hour after water gavage (135 c.c.) and remained there until the baby's discharge from the hospital on the twelfth day. Coincident with the drop in temperature there was a fall in the serum protein concentration from 8.06 to 7.09 per cent., and in the red cell count from 5,070,000 per cubic millimeter to 4,030,000.

The Effect of Fluid by Hypodermoclysis and Intraperitoneal Injection on the Temperature and Blood Concentration of the New-born with Fever.—When fluid was given hypodermically or intraperitoneally in amounts varying from 35 to 60 c.c. per kilogram of body weight the effect on the temperature and blood concentration was neither so marked nor so prompt as after water gavage, and occurred much less regularly. Usually a gradual drop of temperature from 0.5 to 1 degree C. in two hours occurred. The drop in the serum

TABLE 1.—*Saline by Hypodermoclysis Without Any Effect on the Temperature and Blood Concentration in a New-Born with Fever*

Time, a. m.	Temperature		Serum Protein, per Cent.	Red Cells, per Cu. Mm.
	Centigrade	Fahrenheit		
9:00	38.7	101.6	....	.....
10:00	39.3	102.8	....	.....
10:30	39.4	103.0	8.13	4,180,000
10:45	39.5	103.2	8.30	.....
11:15	39.9	103.8	....	.....
11:25-11:45 hypodermoclysis, 125 c.c. saline				
11:50	39.7	103.5	....	.....
12:00	39.7	103.5	7.78	.....
p. m.				
12:15	39.8	103.7	7.78	4,180,000
12:40	39.8	103.7	....	.....
1:10	39.8	103.7	7.84	.....
1:30	39.5	103.2	7.84	.....
2:00	39.5	103.2	....	.....
3:00	40.0	104.0	....	.....
3:30	40.0	104.0	7.84	.....
3:30-4:00 colon irrigation				
4:25	39.4	103.0	7.61	.....
4:55	39.3	102.8	7.73	.....

TABLE 2.—*Saline by Hypodermoclysis with an Immediate Fall in Temperature and Blood Concentration Followed by a Secondary Rise in a New-Born with Fever*

Time, p. m.	Temperature		Serum Protein, per Cent.
	Centigrade	Fahrenheit	
3:00	39.3	102.8	7.55
3:10	39.3	102.8	7.55
3:25	39.2	102.6	....
3:45	39.3	102.8	....
4:25	39.0	102.2	8.02
4:40-4:50, hypodermoclysis, 150 saline			
5:00	38.6	101.4	....
5:10	38.4	101.1	7.73
5:20	38.2	100.8	....
5:40	38.1	100.6	7.55
5:50	37.9	100.2	....
6:30	38.1	100.6	7.37
7:45	39.2	102.6	7.55
9:05	39.2	102.6	....
9:10	39.4	103.0	7.96
9:40	39.3	102.8	....
10:15	39.5	103.2	7.78

TABLE 3.—*Saline by Intraperitoneal Injection Followed by a Slight Transitory Drop in Temperature and Blood Concentration in a New-Born with Fever*

Time, a. m.	Temperature		Serum Protein, per Cent.
	Centigrade	Fahrenheit	
10:00	38.9	102.0	7.76
10:10	38.7	101.6	....
10:25	39.0	102.2	7.95
10:30	39.0	102.2	....
10:45	38.8	101.8	7.61
11:00-11:15, intraperitoneal injection 150 c.c. saline			
11:20	38.7	101.6	7.67
11:25	38.1	100.4	....
11:30	38.6	101.4	....
11:35	38.3	101.0	7.61
12:00	38.5	101.2	7.55
p. m.			
12:15	38.6	101.4	....
12:25	38.6	101.4	7.61
12:55	38.6	101.4	....
1:35	38.4	101.0	7.96

protein concentration rarely amounted to over 0.5 per cent. of protein. In only three of the sixteen babies studied after fluid was given by these routes did the temperature reach normal during the period of observation, which was usually about two hours after the fluid was given. The results in four cases are shown in Tables 1 to 4.

TABLE 4.—*Saline by Intraperitoneal Injection, with a Gradual Fall in Temperature and Blood Concentration in a New-Born with Fever*

Time, a. m.	Temperature		Serum Protein, per Cent.
	Centigrade	Fahrenheit	
10:00	39.7	103.4	7.75
10:30	39.7	103.4	7.61
10:30-10:45, intraperitoneal injection 150 c.c. saline			
10:50	39.4	103.0	7.96
10:55	39.3	102.8	7.61
11:10	39.0	102.2	....
11:15	38.8	101.8	....
11:40	38.6	101.4	7.43
12:00	38.3	101.0	7.55
p. m.			
12:30	38.7	101.6	....
1:00	38.7	101.6	7.49
1:45	38.8	101.8	....

TABLE 5.—*The Effect of Water by Gavage on the Temperature and Blood Concentration of Infants with Fever Due to Various Infections*

Case	Age, Mo.	Weight, Kg.	Diagnosis	Amount of Fluid Given, C.c.	Temperature				Serum Protein, per Cent.		Time Between the Beginning of the Experiment and the Maximum Drop in Temperature
					At the Beginning of the Experiment		At the Time of the Maximum Drop		At the Beginning of the Experiment	At the Time of the Maximum Drop in Temperature	
					F.	C.	F.	C.			
1	9	4.15	Otitis media	210	102.2	39.0	104.2	40.1	7.17	....	2 hrs.
2	10	5.81	Acute bronchopneumonia	150	102.2	39.0	100.8	38.1	8.83	....	2 hrs.
3	15	4.90	Acute bronchopneumonia	210	102.0	38.9	101.0	38.2	7.69	....	2 hrs.
4	3	3.38	Otitis media	150	102.0	38.9	102.8	39.4	5.39	....	2 hrs.
5	10	7.84	Nasopharyngitis	240	102.0	38.9	101.6	38.7	....	....	1½ hrs.
6	4	3.75	Miliary tuberculosis	210	102.0	38.9	101.4	38.6	5.81	5.75	1 hr.
7	8	5.63	Acute bronchopneumonia	180	100.8	38.1	100.4	38.0	7.50	7.09	1 hr.
8	2	3.92	Nasopharyngitis	150	100.8	38.1	100.4	38.0	6.91	6.51	2 hrs.
9	4	3.68	Miliary tuberculosis	180	104.4	40.2	101.0	38.2	6.11	5.87	2 hrs.
10	7	3.83	Nasopharyngitis	150	101.4	38.6	99.6	37.6	5.99	5.99	1 hr.
11	9	6.36	Miliary tuberculosis	225	102.4	39.2	101.6	38.7	7.20	6.91	1½ hrs.

The Effect of Fluid by Mouth on the Temperature and Blood Concentration of Infants with Fever Due to Various Infections.—Water was given by gavage to eleven infants with fever due to various infections. The results are shown in Table 5. In most instances there was a slight fall in the temperature amounting to less than 1 degree C.

In one infant with nasopharyngitis and with a temperature of 38.1 C. (101.4 F.) the temperature dropped to 37.6 C. (99.6 F.) in one hour. The plasma dilution following the water gavage in these older infants with fever was not so marked as in new-born infants with fever.

COMMENT

These results emphasize two points of interest. First, the striking parallelism between the effect of fluid on the blood concentration and on the temperature in the new-born with fever offers strong evidence in favor of the view that this particular type of fever is due to dehydration. A second point of interest is the greater efficacy of the oral over the subcutaneous or intraperitoneal routes in reducing the concentration of the blood and lowering the temperature in the new-born with dehydration. When the fluid is given by mouth it enters the blood promptly, as is indicated by a fall in the serum protein concentration and in the red cell count. When the fluid is given hypodermically or intraperitoneally the fall in blood concentration is neither so marked nor so prompt as after gavage. Apparently a process exists whereby fluid given by mouth is promptly absorbed into the blood and tissues. Fluid given into the subcutaneous tissues or the peritoneal cavity enters the blood much more slowly and to a much less extent.

Similar results were obtained by one of us (B.) in older infants dehydrated as a result of severe diarrhea. During the summer of 1921 a series of these infants was treated with fluid by mouth. Water or 5 per cent. glucose solution was dripped into the stomach at a slow rate by means of a tube inserted into the stomach through the nose. A fine catheter (No. 8 French), well lubricated, was used and the fluid administration continued for several hours. A drop in blood concentration, an improvement in the skin turgor and an increase in the urine flow followed when fluid was given in this manner. When the fluid was given hypodermically or intraperitoneally the increase in plasma water was slight or entirely absent and there was usually no effect on the urinary flow. An improvement in the skin turgor in the neighborhood of the fluid injection was frequently observed.

It is of interest to note in this connection that Robertson and Bock [4] found that, in adults with post-hemorrhagic anemia, fluid by mouth was second only to blood transfusion in the rapidity with which it increased the blood volume.

SUMMARY AND CONCLUSIONS

1. The effect of fluid on the temperature and blood concentration of the new-born with fever was studied.

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4. Robertson, O. H., and Bock, A. V.: The Use of Forced Fluids by the Alimentary Tract in the Restoration of Blood Volume After Hemorrhage, J. Exp. Med. **29**:155 (Feb.) 1919.

2. When fluid was given by mouth in the proportion of from 30 to 40 c.c. per kilogram of body weight a prompt fall in the temperature and blood concentration followed with great regularity. The temperature usually reached normal within from 30 to 90 minutes.

3. When the fluid was given hypodermically or intraperitoneally, the effect on the temperature and blood concentration was neither so marked nor so regular as after water by mouth.

4. Water by mouth, in amounts similar to those used in the newborn with fever, has practically no effect on the temperature of infants with fever due to various infections.

5. In a group of older infants, dehydrated as a result of severe diarrhea, fluid by mouth was much more effective in reducing the concentration of the blood than fluid given by hypodermoclysis or intraperitoneal injection.

REPORT OF CASES

CASE 1 (Table 1).—The baby, a girl, was delivered normally at term and weighed 3.21 kg. On the third day, after a loss of weight of 300 gm., she developed fever. The serum protein concentration was 8.30 per cent., indicating a marked diminution in plasma water. A subcutaneous injection of saline was given. There was no change in the temperature or red cell count. The serum proteins fell slightly. The fever persisted for twenty-four hours, after which time it fell to normal and remained there until the baby's discharge from the hospital, which occurred on the eleventh day.

CASE 2 (Table 2).—The baby, a boy, was vigorous at birth and weighed 4.50 kg. The protein concentration of the placental serum was 7.61 per cent., indicating a slight diminution in the plasma water. He developed fever on the second day after a loss of weight of 450 gm. He was somnolent and limp and cried very little. The skin was dry and inelastic, the mucous membranes of the mouth dry and red. The serum proteins had risen to 8.02 per cent. A subcutaneous injection of saline was given. A slight fall in temperature and blood concentration occurred during the ninety minutes after the hypodermoclysis but both rose again quickly. The temperature remained elevated for three days, after which it dropped to normal and remained there until the baby's discharge from the hospital.

CASE 3 (Table 3).—The baby, a boy, was delivered normally and weighed 3.84 kg. He developed fever on the second day after a loss of 90 gm. The serum proteins were 7.96 per cent. The skin was dry and inelastic, the mouth and lips dry. An intraperitoneal saline injection was given. There was a slight fall in temperature and blood concentration during the forty-five minutes after the injection followed by a secondary rise.

CASE 4 (Table 4).—The baby, a girl, developed fever on the second day after birth, after a loss of 330 gm. The next day she had lost an additional 240 gm. and the temperature rose to 40 C. (104 F.). She was somnolent and limp and appeared ill. The skin was moderately inelastic, the mouth dry and red. She took the breast eagerly. The serum proteins were 7.78 per cent. An intraperitoneal injection of saline was given. There was a slight drop in temperature and serum concentration. Fever persisted for another day after which the baby was afebrile except for a slight rise in temperature, associated with a loss in weight and an increase in the serum protein concentration, on the eleventh day.

TUBERCULOUS PULMONARY CAVITIES IN INFANCY*

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Tuberculous pulmonary cavities in infancy would seem at first glance to be a very rare condition. A review of the available literature demonstrated, as it so often has done in the past, that this occurrence, while indeed uncommon, is not at all rare. The French literature, especially, supplies numerous instances of cavity formation following tuberculous infection. Our own literature is surprisingly meager on this subject. The purpose of this paper is the presentation in detail of a case showing several unusual and interesting features. A review of available case reports is submitted.

REVIEW OF LITERATURE

In 1888, Leroux,[1] in a study of 219 infants from birth to 2 years of age, was able to demonstrate cavities in fifty-seven cases. Twelve cases were under 6 months of age and five cases were under 3 months.

In 1897, Babeau [2] reported a case of cavity formation in an infant 10 months old. Later in the same year, Ausset [6] reported a cavity in an infant 14 months old.

Comby,[3] in 1898, found seven cases of cavity formation in twenty infants dying of tuberculosis.

Kuss [4] and d'Aviragnet [5] in extensive Paris theses published during the same year, summarized all the available French literature. They found that cavities occurred with surprising frequency. During the first year of life one out of every five patients dying of pulmonary tuberculosis showed evidence of cavity formation.

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* Received for publication, Dec. 20, 1923.

* From the department of pediatrics, University of Minnesota.

* Read before the medical staff of the Lymanhurst School for Tuberculosis Children, May 22, 1923.

1. Leroux: La tuberculose du premier âge. Études expérimentales et cliniques sur la tuberculose, Paris, 1888, p. 2.

2. Babeau, Joseph: Tuberculose généralise caverne pulmonaire chez une enfant de dix mois, J. de clin. et de thérap. inf., Paris, **5**:483-487, 1897.

3. Comby: Tuberculose pulmonaire chez l'enfant, Arch. d. mal. des enfants, 1898.

4. Kuss: Tuberculose cavitaire en un enfant, Thèse de Paris, 1898.

5. D'Aviragnet: Tuberculose cavitaire chez un enfant, Thèse de Paris, 1898.

In 1899, Ausset [6] reported a tuberculous cavity in an infant aged 11 months. During the same year, Griffon [7] reported a cavity in an infant 6 months old.

Rabot and Varay,[8] in 1902, described the necropsy on an infant 5 months old. The right lung showed two cavities, the size of a hazel nut and a pea, respectively. The left lung showed a cavity the size of a large pea. Mayound,[9] at the same time, reported a cavity in an infant 3 months old.

In 1903, Hautant [10] cited a case in an infant aged 9 months. In the same year, Mairesse,[11] in his Paris thesis, reviewed thirty-five cases in infants ranging from 2 months to 2 years of age. He concluded that cavitation in early infancy was rare and they were exceptional before the first year. He thought they were due to a rapid "melting of a caseous pneumonia."

In 1905, Delarde [12] reported the case of a 10 months infant.

Lortat and Vitry,[13] in 1905, demonstrated a cavity the size of a grain of hempseed in an infant 2 months old.

Geipel,[14] in 1906, justly emphasized a critical review of the reported cases. In thirty-four cases of tuberculosis in infancy, he found thirteen cases of cavity. Later, in 1906, Koch-Elberfeld [15] published a case of a cavity formation in a child aged 5 months.

About the same time, in 1906, L'Homme,[16] in a thorough research, reviewed thirty-nine cases collected from the literature (including three of his own). His patients varied in age from birth to 24 months. He

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6. Ausset, M.: Tuberculose cavitaire, en un enfant de onze mois, Rev. mens. d. mal. d'l'enf. **17**:332, 1899; Centr. Med. Soc. du Nord, May 12, 1899.

7. Griffon, M. V.: Phlébite oblitérant de la vein cave inférieure dans un cas de tuberculose cavitaire de la premier enfance, Rev. Mens. d. mal. de l'enf. **17**:30, 1899.

8. Rabot, and Varay, F.: Tuberculose pulmonaire ulcéreuse (cavernes) chez un enfant de cinq mois, méningite tuberculeuse (forme hémiplégique), Rev. Mens. de mal. de l'enf. **20**:246-254, 1902.

9. Mayound: Caverne pulmonaire chez un nourrisson, Lyon Méd., **98**:21-22, 1902.

10. Hautant, M. A.: Tuberculose pulmonaire avec grande caverne, chez un enfant de neuf mois, Tubercul. Inf., Paris **6**:83-87, 1903-1906.

11. Mairesse, Marcel: Des cavernes pulmonaires tuberculeuse dans le premier âge, Paris Thèses, 1903-1904.

12. Delarde, M.: Caverne pulmonaire chez un enfant de dix mois, Echo méd. du nord, Lille **9**:606, 1905.

13. Lortat, Jacob, and Vitry, G.: Caverne pulmonaire chez un nourrisson, Cong. Interat. de la tuberc., Par. **2**:350-355, 1905.

14. Geipel, P.: Ueber Sauglings-tuberkulose, Ztschr. f. Hyg. u. Infections-frankh., Bd. **53**:51-58, 1906.

15. Koch-Elberfeld, Max: Kavernose Lungenphisis bei einem fünf Monate alten Kind, Verhandl. d. deutsch. path. Gesellsch. **10**:262-264, 1906.

16. L'Homme, Henri: Contribution a l'étude de la tuberculose chez le nourrisson dans la premier enfance, Paris Thèses, 1905-1906.

quoted Huguenin [17] as reporting a case of cavity formation in a newborn infant. He also cited Berti's [18] case of lung cavity in an infant 9 days old. From his studies he concluded that cavity formation was uncommon but not rare and that its clinical diagnosis was exceedingly difficult. He thought that the prognosis was not always fatal, but that it usually was fatal.

Monges,[19] in 1907, published a report showing cavity formation in an infant 4½ months old. Sauvan [20] reported, in the same year, a tuberculous cavity in a 13 months old infant.

Fig. 1.—Left: Marked density along the hilum and root of right lung and irregular density of its entire upper lobe, a suggestion of a large anular shadow between first and third ribs near hilum, giving the impression of a resolving lobar or bronchopneumonia. (The roentgenogram is reversed.) Right: Right lung field showing irregular density throughout, except in apex and base; a large annular shadow within this dense area in second and third interspaces near hilum, which simulates a cavity. The general appearance of the lungs has not changed materially, except for a greater prominence of the annular shadow. The impression given was probable of a pulmonary tuberculosis with irregular consolidation and a large cavity in the right upper lobe.

White and Carpenter,[21] in 1909, in an excellent presentation of the subject, reported six cases of cavity formation, occurring in colored infants between 5 and 15 months old. In every instance the diagnosis was made at necropsy.

17. Huguenin: Tuberculose des nouveau-né et tuberculose congénital, Gaz. d. Hôp. **61**:785, 1888.

18. Berti, G.: Interus ala possibilite di processi tisiogeni congeniti, Bol. del. scien. med. **10**:29, 1882.

19. Monges, M. J.: Cavernes tuberculeus chez un enfant de quatre mois et demi, Marseille méd. **44**:153, 1907.

20. Sauvan, M.: Caverne tuberculouse chez un enfant de treizième mois, Marseille méd. **44**:343, 1907.

21. White, C. Y.; and Carpenter, H. C.: Tuberculose Pulmonary Cavities in Infants, Am. J. M. Sc. **138**:79-92 (July) 1909.

During the same year, Gaujoux and Brunel [22] collected forty-one cases from the French literature, and in a very thorough and careful study presented their results. There were only twenty-four cases showing cavitation up to 2 years of age. They emphasized the difficulties of clinical diagnosis and the importance of a critical study of cases reported as showing cavitation.

Chalier,[23] also in 1909, presented a patient 1½ years old.

In 1910, Collet and Delachanal [24] cited the case of a 3 months old infant in which they found one cavity at the apex of the right lung, and another in the lower lobe. They thought that cavities in babies were formed by a rapid caseation necrosis of lung tissue and not by a fibrocaseous process as in the adult.

Buddy,[25] in 1911, reported a baby girl, 3 months old, showing an abscess cavity.

Ribadeau-Dumas,[26] in 1913, laid emphasis on the value of the roentgen ray in the diagnosis of tuberculous cavities. Clinical findings were unreliable because of the small size of the lesions. He found that they varied from that of a nut to that of a pea. He thought that it was due to this factor that the condition is found so rarely.

In 1914, Wollstein and Bartlett,[27] in an extensive study of tuberculous lesions in infants and young children based on necropsy examinations, drew some interesting conclusions. In 178 necropsies on tuberculous children, pulmonary cavitation was present in forty-three. The right lung was more frequently involved than the left. Twenty patients were less than 1 year old and the youngest was 3¾ months. They found that "cheesy degeneration in the lungs of these young children takes place and extends rapidly."

In 1915, Randolph [28] reported a case of cavitation of the lung in an infant dying in the seventh month.

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22. Gaujoux, E., et Brunel: La tuberculose pulmonaire caverneuse chez L'enfant, Ann. de méd. et chir. inf. **13**:397-401, 443, 472, 1909.

23. Chalier, M. J.: Tuberculose cavitaire de la base, greffec sur un ancien, Foyer de bronchopneumonie, chez un enfant de un an et demi, Lyon méd. **113**: 743-745, 1909.

24. Collet and Delachanal: Cavernes tuberculeuse chez un nourrisson de trois mois, Lyon Méd. **115**:317-321, 1910.

25. Buddy, E. P.: Report of a Case of Pulmonary Tuberculosis with Cavity Formation in an Infant Aged Three Months, J. Missouri M. A. **8**:359-360, 1911-1912.

26. Ribadeau-Dumas: Tuberculose cavitaire du premier âge, Bull. et. mém. Soc. méd. d. hôp. de Paris **36**:936-942, 1913.

27. Wollstein, Martha, and Bartlett, F. H.: A Study of Tuberculous Lesions in Infants and Young Children, Based on Postmortem Examinations, Am. J. Dis. Child. **8**:362-377 (Nov.) 1914.

28. Randolph, B. M.: Case of Cavitation of Lung in an Infant Dying in the Seventh Month, Washington M. Ann. **13**:149-150, 1914-1915.

Bergmann,[29] in the same year, published a case report of an infant 82 days old.

Canelli,[30] in 1919, found twenty-five cases of cavernous tuberculosis in 175 patients dead of pulmonary infection.

In the second 6 months of life true cavities were seen in four instances.

Ribadeau-Dumas is quoted as saying that he found cavernous pulmonary tuberculosis in 32.4 per cent. of cases during the first 2 years of life.

In 1921, Schott[31] reported multiple cavities in an infant aged 9 months. The cavities were of the honeycomb type, of good size, sharply limited and with smooth walls.

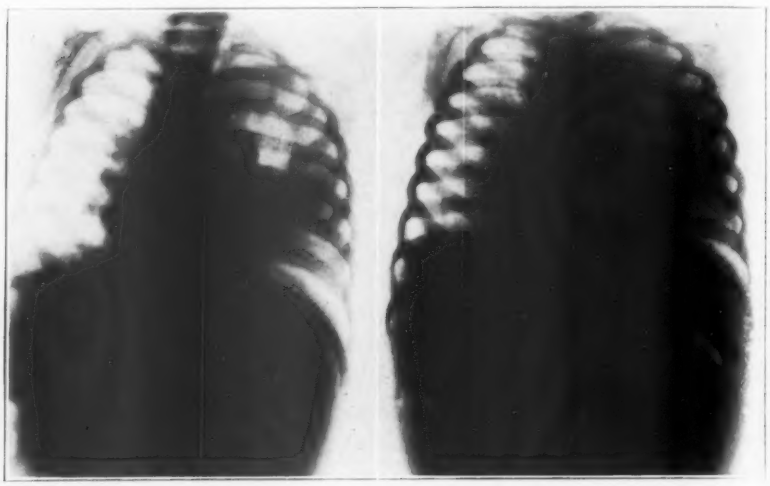

Fig. 2.—Left: The appearance of the right lung is about the same as in Figure 1. The cavity now appears to be well defined. Right: The appearance of the chest is about the same as Figure 1. The cavity is apparently the same size. The consolidation appears to be more homogeneous in density and has extended into the periphery.

REPORT OF CASE

History.—A boy, colored, aged 2 years, was admitted to hospital, Dec. 6, 1922, with the complaint of shortness of breath, fever, loss of appetite, and cough of one month's duration.

Past History.—The patient was born at full term after a normal delivery, and weighed 8 pounds (3,628 gm.) at birth, he was breast fed for five months.

29. Bergmann, H.: Cavernöse Lungentuberkulose beim Säugling, Berl. klin. Wchnschr. **52**:577-579, 1915.

30. Canelli, Adolfo: Contributo alla conoscenza delle localizzazioni della tubercolosinelli primae eta, Riv. di. clin., pediat., Firenze, **17**:523, 1919.

31. Schott, A.: Ueber eine eigenartige tuberkulose Wabenlungen in Säuglingsalter, Beitr. z. Klin. d. Tuberk. **48**:112 (June 10) 1921.

He had no diseases, except a severe attack of pertussis in July. There was no history of tuberculosis in the family or no exposure to tuberculosis.

Physical Examination.—On admission the temperature was 104; the pulse 120. The patient was well developed, fairly well nourished, irritable and restless. The eyes, ears, nose and throat were negative. The heart tones were rapid but regular. The lungs had an impaired resonance over the entire right side posteriorly with numerous moist râles heard throughout this area. Moist râles were also present in the left lower lobe. Tubular breathing was present over the right upper lobe posteriorly. The liver and spleen were palpable 4 cm. below the costal margin.

Laboratory Findings.—Urine, amber, acid, specific gravity, quantity not sufficient; albumin, negative; pus cells, 2 per high power field; blood, hemoglobin 70 per cent.; red blood counts, 4,400,000, leukocytes, 15,750; polymorphonuclears, 89 per cent.; lymphocytes, 11 per cent.; Wassermann test, negative repeatedly. the Pirquet and Mantaux tests were positive; no tubercle bacilli were demonstrated in the feces.

Fig. 3.—Right lung, weight 310 gm., showing thickened adherent pleura, minute miliary nodules and a large cavity containing a large amount of pus. The cavity, located at the junction of the medial and post parts of the upper lobe, measured 5 by 4.5 cm.

On December 14, the patient showed no charge in condition. He had a temperature varying from 99 to 103 daily. The physical findings in the chest increased and were apparently limited to the right side. December 30, the chest plates became more suggestive of possible cavity formation. There was no change in the physical findings. Jan. 10, 1922, the condition remained the same. February 4, the patient lost weight in the last week, and was failing very rapidly. There was no evidence of meningitic involvement. The patient died on February 5, at 8:50 a. m.

Necropsy.—This was limited to the chest. We are indebted to Dr. J. Wendell Gullikson for the pathologic report.

The body was that of a well developed, but emaciated colored child, measuring 85 cm. in length. There was an abscess in the left ischiorectal fossa.

The pleural cavities were obliterated by fibrinous adhesions. The pericardial cavity contained about 25 c.c. of clear straw colored fluid. The liver extended 8 cm. below the root of the xiphoid and 8 cm. below the costal margin in the midclavicular line, almost down to the anterior superior spine of the ilium.

The heart weighed 16 gm. The muscle was slightly pale; otherwise the heart was normal. The root of the aorta was normal. The left lung weighed 150 gm. It was tightly adherent to the thoracic wall by fibrinous and fibrous adhesions. Crepitation was markedly reduced. There was a whitish nodule under the pleura measuring 2 by 1 cm. This nodule was located near the lower part of the lateral surface of the lower lobe. On section this nodule appeared to be quite firm. Section of the remainder of the lower lobe showed an increased amount of frothy fluid with some pus expressible in some dense areas.

The right lung weighed 310 gm. It was firmly bound down with fibrinous adhesions anteriorly and laterally and by fibrinous adhesions posteriorly. Manipulation of the right lung in removing it ruptured a cavity and much pus escaped. The cavity was located at the junction of the medial and posterior parts of the upper lobe and measured 4 by 5 cm. There was a small cavity measuring 1.5 cm. in diameter near the apex. The lower part of the lower lobe was infiltrated with smaller whitish nodules which on section were harder than the normal lung tissue. Pus was also expressible from the bronchi.

Fig. 4.—Spleen, weight 130 gm. The capsule was tense and literally covered with whitish nodules, varying in size from a pin point to 5 mm. in diameter.

The spleen weighed 130 gm. Its capsule was tense and was literally covered with whitish nodules which appeared to be in the substance of the organ, and which varied in size from a pin point to 5 mm. in diameter. The left kidney weighed 60 gm. The vessels on the surface were slightly congested. Section showed some pallor of the pulp. Section of the liver showed a few small whitish nodules with a yellowish tinge. The mesenteric lymph nodes were greatly enlarged and some of them measured up to 1.5 cm. in length. They were very hard to the touch. Some showed evidence of caseation. The peribronchial lymph nodes were also greatly enlarged and showed some caseation on section.

Diagnosis.—The final diagnosis was: (1) Pulmonary tuberculosis with cavities in the right lung; (2) miliary tuberculosis of spleen and liver; (3) tuberculous adenitis; (4) slight bronchopneumonia; (5) fibrinous and fibrous pleuritis.

COMMENT

A review of the literature on cavity formation during the first 2 years of life is unsatisfactory. In many instances, particularly in the French literature, the age of the patient is not given, and only a very cursory description of the findings are submitted.

The frequency of occurrence of pulmonary cavitation is uncertain. Apparently the results are dependent on the observer and on the locality. While some men find cavities very common, others still consider them rare. Garrod, Batten and Thursfield,[32] in speaking of tuberculosis in early life, say, "Well marked, cavernous signs are seldom present and consequently it is sometimes said that cavities are rare in early life. This, however, is incorrect; they are frequently seen on the necropsy table."

Schlossman [33] holds the same opinion. On the other hand, Smith,[34] in a recent paper, reports statistics on 200 consecutive necropsies from the Infants Hospital of Boston, showing an incidence of 20 per cent. tuberculous. The distribution of the lesions is discussed but apparently no cavity formation was found.

Morse [35] recently made the following statement: "In my experience, cavities in the lungs in childhood are very seldom due to tuberculosis, because children, as a rule, die before cavities have formed."

Our pediatric textbooks dismiss the subject with the statement that cavities may occur at any age.

The subject is one of considerable theoretic and some practical interest. More case reports and thorough discussions are desirable.

The case we report is of interest because the clinical findings suggesting cavity formation were confirmed by our roentgen-ray studies. A diagnosis of pulmonary cavity of tuberculous origin was made during the life of the infant. This is the first reported case, so far as we know, in which the diagnosis made prior to necropsy was confirmed later. The prolonged period of observation gave an excellent opportunity for careful clinical and roentgen-ray study, showing the progressive formation of the cavity.

CONCLUSIONS

1. A review of available literature concerning pulmonary cavitation of tuberculous origin in infants up to 2 years of age, with special regard to incidence, is submitted.

2. A case of tuberculous pulmonary cavity in an infant 2 years of age is confirmed at necropsy.

3. Tuberculous cavities in infants are the result of caseous pneumonia, and not of chronic ulcerative tuberculosis as in adults.

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32. Garrod, A. E.; Batten, F. E., and Thursfield: Diseases of Children, Tuberculous Cavities, London, 1913, p. 1116.

33. Schlossman: Ueber Tuberculose, Berl. klin. Wchnschr., Oct. 6, 1902, p. 951.

34. Smith, L. W.: Tuberculosis in Infancy and Childhood, Am. Rev. Tuberc. **6**:201-212 (May) 1922.

35. Morse, J. L.: Tuberculosis in Children from the Standpoint of the Pediatrist, Boston M. & S. J. **187**:463-466 (Sept. 28) 1922.

REPORT OF A CASE OF ECTOPIA CORDIS*

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Cases of ectopia cordis are sufficiently infrequent to warrant recording the following one.

REPORT OF CASE

Mrs. P. S., an Irish American, aged 43, a primagravida, first came under observation Oct. 5, 1923. The patient's family and personal history were entirely negative. She had been married less than a year at this time. Her husband had had a healthy child by a previous marriage and appeared to be in excellent health himself. The patient began to menstruate at the age of 15, which lasted for from four to five days' at twenty-eight day intervals, and was moderately profuse all her life, more so during the two years prior to the time she first came under observation; she never had any pain with menstruation. The last menstruation had occurred on April 3, quickening was noticed August 25, and the date of the expected birth was about Jan. 10, 1924.

The patient presented a blood pressure of 140 systolic, 90 diastolic, the urine was entirely negative, with a specific gravity of 1.014. Up to November 16, the blood pressure on four different occasions varied from 140 to 154 systolic, 88 to 90 diastolic. On October 27, the urine first showed a trace of albumin, specific gravity 1.023; there was no other evidence of renal involvement. Several other specimens gave the same analytic results. She was admitted to the hospital for observation on November 17, when she presented marked swelling of the feet and ankles, with some headache and visual disturbances.

Physical Examination.—The scalp and head were normal; the skin normal; the eyes reacted to light and in accommodation; the conjunctivae were pale; the extra ocular movements were normal; the ears and nose were normal; the mouth showed a degree of gingivitis; the teeth were fairly well cared for; the pharynx was slightly congested; the tongue was clean; the muscular movements were normal. The neck showed no adenopathy. There were visible pulsations of the great vessels in the neck. There was no palpable enlargement of the thyroid. The lungs were normal except for a few scattered râles at the bases posteriorly. The point of maximum impulse of the heart was in the fifth interspace, 3½ inches (8 cm.) from the midline; the sounds at the apex were regular, of fair muscular quality with no murmurs; at the right base a blowing diastolic murmur was heard, transmitted to the left and distinctly heard in the fourth interspace at the border of the sternum. The abdomen was uniformly distended and tense; the fetal parts were palpable, but no fetal heart sounds could be made out. The extremities were markedly edematous below the knees.

Roentgenogram showed a shadow of the fetal skeleton with the skull in the upper left quadrant.

On November 21, the urine showed a specific gravity of 1.020, a trace of albumin, a large number of pus cells, epithelial cells and a few granular and hyaline casts. On November 22, the specific gravity was 1.018, with increasing albumin, a few pus cells and a few granular and hyaline casts.

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* Received for publication, April 2, 1924.

* From the Laboratory and Obstetrical Division of the Jersey City Hospital, Jersey City, N. J.

Blood pressure range: November 19, 130 systolic, 70 diastolic; November 22, 140 systolic, 90 diastolic; November 24, 142 systolic, 86 diastolic; November 26, 150 systolic, 88 diastolic.

The pulse was from 100 to 120; the temperature was normal; the respirations from 24 to 34.

Renal function test, November 18, showed 10 per cent. the first hour and 15 per cent. the second hour.

The blood chemistry on November 19 showed nonprotein nitrogen 32 mg. per hundred cubic centimeters of blood, urea 12 mg., creatinin 1.7 mg., sugar 76 mg.

Fig. 1.—The body of the distorted fetus.

On November 26, at 5 p. m., labor was induced by the insertion of a Number 4 Voorhees bag, as the indication was the presumptive death of the fetus in utero and increasing toxemia of the patient, in spite of hospitalization and treatment. At 10:30 a. m. the following day, delivery of a stillborn fetus was effected by median forceps application.

The patient's convalescence was uneventful, the blood pressure gradually falling and the subjective evidences of toxemia entirely disappearing. The patient was discharged from the hospital on December 13.

Necropsy (November 27, 3 p. m.).—The body was that of a very much distorted and somewhat macerated fetus, measuring 37.5 cm. in length and weighing about 4½ pounds (2,041 gm.). There was an extensive swelling, reaching from the lower jaw around the neck and downward over the sternum, most marked toward the left side, and giving the appearance of the head attached

directly to the thorax, without an intervening neck. The skin of the entire body was reddish, most pronounced in the region of the head and neck, and the epidermis was peeled off in part. The heart and a portion of its vessels lay free on the chest wall. The forearms were tightly flexed on the arms, appearing as marked contractures, and the ventral surfaces of the hands were whitish, softened and partially macerated. The left ear presented a similar but more advanced macerated appearance. About 2 cm. of umbilical stump was still attached. The fontanels were depressed and the cranial sutures approximated. The eyes and ears were normal. The vault of the mouth contained a complete fissure, exposing the nasal fossae. The heart was found lying in a bed formed by a depression in the chest. Both lower extremities showed an equinovarus. The diaphragm reached the fourth rib on both sides. The scrotum was markedly swollen.

The peritoneal cavity was free from fluid and adhesions. The intestines were collapsed and, on examination, contained only a small quantity of mucus; they were patent. The rectus muscles were somewhat diastatic, but there was no distinct epigastric hernia. The adrenals showed no gross lesions. The

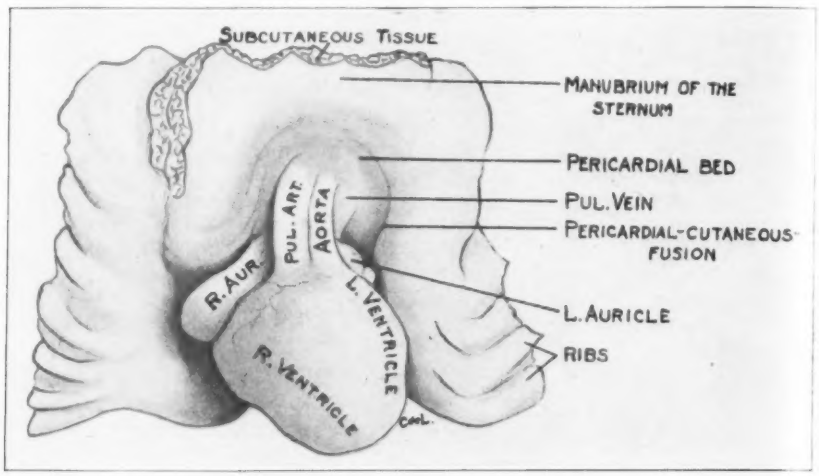

Fig. 2.—The heart as presented on the chest, showing anatomic details.

kidneys were lobulated, but otherwise grossly normal; the pelves and ureters were normal. The bladder was contracted and smooth. The pancreas, spleen and liver showed no gross lesions. Both testicles were in the inguinal canal.

The ventral thoracic wall, lungs and heart were removed en masse. The lungs on the outer surfaces showed some atelectasis; the pleural cavities were normal. Some thymic remains were found behind the pericardial membrane. The heart was found lying in a bed formed by the depression caused by the fissured sternum, which was lined by pericardial membrane. The pericardium, as it advanced anteriorly, was apparently fused with the skin of the anterior chest wall. The sternum was fissured, producing a gaping orifice through which the heart protruded. In its manubrial portion, the sternum was represented as a single bone. Examination of the heart by incising the four chambers showed them to be distinct cavities, with patent valvular orifices. The coronary vessels could not be examined. The pulmonary artery and aorta, though joined together at their commencement for a distance of about 1.5 cm., were nevertheless on incision and exploration with a probe, found to be two distinct tubes. Patency of the ductus arteriosus and foramen ovale could not be determined without destroying the specimen.

The tongue, trachea and thyroid were removed with the thoracic organs, but showed no gross abnormalities. The head was grossly normal.

COMMENT

That ectopia cordis is a rare condition is testified by the meager literature containing isolated case reports. Its relative frequency cannot be given with any approach to accuracy. Thus, during twenty years in the necropsy rooms of Bellevue Hospital, not a single case was encountered. Neither have we been able to find statistical literature in other hospital reports. In Allbutt's [1] *System of Medicine* mention is made of the rarity of the three forms of the lesion noted by Rauchfuss, namely, ectopia cordis abdominalis, cervicalis and pectoralis. Of the three, the last is said to be the most frequently encountered, and the cervical form is said to be incompatible with life. In cases where the heart is enclosed in a pericardial sac, life may continue for days; those without a sac do not live. Recently, Holmes [2] reported a case in an infant from Howland's Clinic which lived for fifteen months. At birth, a "raw" and bright red pulsating mass was found in the epigastrium. After four weeks, with constant dressing, the mass became covered by skin. Cases have been observed in this country by Holt, Jacobi and Welch. There is an excellent article with a case report by Ellis.[3] Abbott [4] gives a brief but very concise discussion of the entire subject in Osler and McCrae's System of Medicine. Ballantyne [5] cited nine cases occurring in the literature.

The lesion is produced by failure of complete development of the sternum. Embryologically, the sternum is formed from bilateral anlages, and failure of the two pieces to fuse causes a fissure sterni with malpositions, principally of the thoracic organs, especially the heart. Schwalbe [6] states that anomalies in the development of the sternum, particularly in the lower animals, is by no means rare and, thus, anomalous positions of the heart, even within the thorax, are frequently produced. It is naturally encountered most often in premature or stillborn infants.

1. Allbutt: System of Medicine, Vol. 6, New York, The Macmillan Company.
2. Holmes, J. B.: Johns Hopkins Hospital Reports **18,** 1917.
3. Ellis: Proc. Path. Soc., Philadelphia, **18**:36, 1906.
4. Abbott, Maude: Osler and McCrae's System of Medicine, Philadelphia, Lea & Febiger, Vol. 4, Ed. 2, p. 346.
5. Ballantyne: Antenatal Pathology, New York, William Wood & Company, 1904.
6. Schwalbe: Morphologie der Missbildungen des Menschen, Jena, Gustav Fischer, 1909, p. 44.

THE SKIN LESIONS IN MENINGOCOCCUS SEPTICEMIA *

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The skin lesions in acute infectious diseases are among the important and most easily determined clinical findings. Their cause, in some instances, has been explained on the basis of the presence of toxic substances, and in other instances it has been shown positively to be due to the presence of microorganisms. The hemorrhagic, purpuric and petechial lesions belong especially to this latter group. The demonstration of typhoid bacilli in the "rose spots," of streptococci in the skin lesions associated with streptococcus septicemia and subacute bacterial endocarditis, of tubercle bacilli in the petechiae or tuberculids seen in acute miliary tuberculosis, are the most striking examples of this type of etiology.

The meningococcemia preceding or associated with acute epidemic cerebrospinal meningitis is manifest in some cases by the appearance of skin lesions of a petechial or purpuric nature. This characteristic has gained the terms "spotted fever," "black fever" and "petechial fever" for the disease. The existence of a meningococcemia in a considerable percentage of meningococcus infections has been demonstrated many times and in the hands of many observers, both by cultures and smears. Andrewes,[1] in 1906, Coles,[2] in 1915, King,[3] in 1918, and many others have shown the organisms in blood smears.

The study of the skin lesions is less frequently reported. Benda,[4] in 1916, studied three cases of meningitis with petechiae, in which he found a purpuric condition, and inflammatory foci with polymorphonuclear infiltration about the arterioles. In one of these cases, the most fulminating in character, he found groups of gram-negative cocci which he thought were meningcocci.

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* Received for publication, April 2, 1924.

* From Department of Pathology, Harvard Medical School, and The Children's Hospital.

1. Andrewes, F. W.: Case of Acute Meningococcal Septicemia, Lancet, **1**:1172, 1906.

2. Coles, A. C.: Cerebrospinal Meningitis; Meningococci Found in Peripheral Blood Films, Lancet **1**:750 (Jan.) 1915.

3. King, W. W.: Early Diagnosis of Cerebrospinal Meningitis by the Examination of Stained Blood Films, J. A. M. A. **71**:2048 (Dec. 21) 1918.

4. Benda, C.: Mikroskopische Befunde in der Haut bei Petechialer Meningokokken-meningitis, Berl. klin. Wchnschr. **53**:449 (April 24) 1916.

Pick,[5] in 1916, demonstrated typical meningococci, either free or intracellular, in the skin arterioles, in the capillaries and perivascular leukocytic infiltration. He also observed the extravasation of red blood cells.

Renault and Cain,[6] in 1920, described the skin lesions in detail. They mentioned marked pericapillaritis, swelling of the endothelial cells,

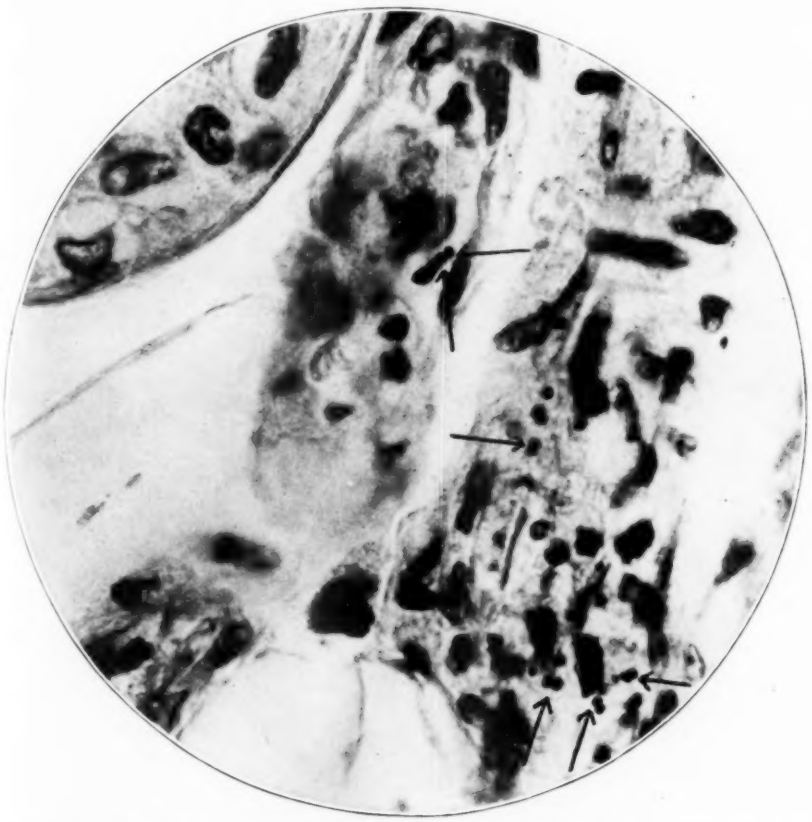

Fig. 1.—Capillaries surrounding coil gland. Several pairs of extracellular meningococci are seen. The arrows point toward the meningococci.

many of which were free in the lumina, together with many polymorphonuclears. In spite of the alteration of the intima there was no tendency to thrombosis. Congestion but not hemorrhage was present. Numerous diplococci were demonstrated in the lesions.

5. Pick, L.: Histiologische und Histiologisch-bacteriologische Befunde beim Petechialen Exanthem der Epidemischen Genickstarre, Deutsch. med. Wchnschr. **42**:994 (Aug. 17) 1916.

6. Renault, J., and Cain, A.: Preuves histologiques et bacteriologiques de l'origine septicémique de purpura de la méningite cérébrospinale, Ann. de méd. **7**:114, 1920.

REPORT OF CASE

The case from which the studies reported here were made, is that of a girl, aged 2 years, who was taken acutely ill, with headache and vomiting of less than two days duration at the time of admission. Physical examination was essentially negative except for a stiff neck, hyperemic nasopharynx, a temperature of 104 F., and a skin condition not unlike a measles eruption over the face and extremities. The trunk was comparatively free of lesions. The eruption rapidly changed to large purpuric spots, and the child died a few hours after

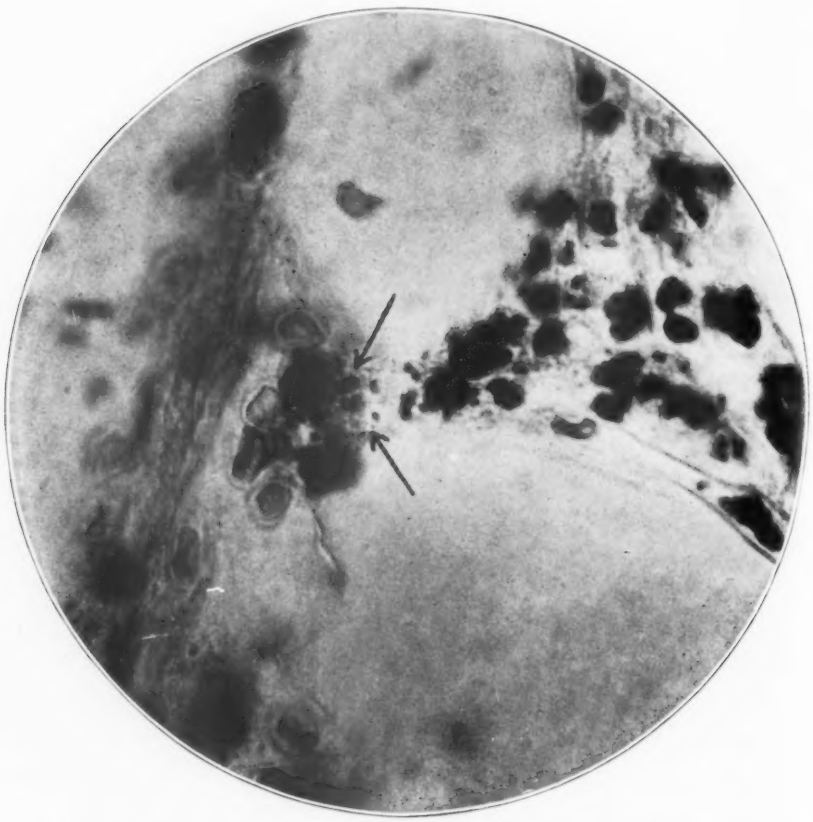

Fig. 2.—Capillaries in subcutaneous stratum. Numerous meningococci are seen in the adjacent fat. Arrow points toward the meningococci.

admission. Blood cultures made during life yielded pure cultures of meningococcus, as proved by morphologic and cultural characteristics supplemented by a positive agglutination reaction with antimeningococcic serum. The spinal fluid was clear and the cell count was not increased.

Restricted necropsy did not permit examination of the central nervous system, but there was no evidence of meningeal involvement by lumbar puncture. The skin lesions were the most remarkable of those found. The epidermis was entirely negative. The arterioles and capillaries in the corium and subcutaneous stratum contained many polymorphonuclears and large mononuclear leukocytes; the endothelial cells were swollen and many were detached

into the lumina. No definite thrombus formation was seen. There was marked inflammatory reaction in the capillaries and small arterioles and perivascular leukocytic infiltration, the infiltrating element being made up largely of polymorphonuclear and large mononuclear leukocytes. The vessels of the subcutaneous stratum (Fig. 2 and 3) and of the capillary bed surrounding the coil glands (Fig. 1) showed the most marked lesions. Especially in the subcutaneous stratum, the vessels were engorged with leukocytes, the vessel walls were densely infiltrated with leukocytes and there was hemorrhage into the adjacent fat tissue. Many intracellular and extracellular gram-negative diplo-

Fig. 3.—Capillaries in subcutaneous stratum, showing engorgement with leukocytes, capillaritis, hemorrhage and numerous intracellular and extracellular meningococci. The arrows point toward the meningococci.

cocci were demonstrated in these areas, as well as in the capillaries about the coil glands, both inside and outside the vessel walls. In a few instances an endothelial leukocyte was observed to have phagocytized one or more pairs of diplococci.

In the other organs the mucosa of the larynx presented the greatest number of organisms. A few were found in the lungs and spleen, but none were found in the liver, kidneys, pancreas, adrenals, gastro-intestinal tract or mesenteric lymph nodes.

The histologic methods used consisted of fixation of the tissue in Zenker's fluid, paraffin imbedding, and staining of the sections by Wolbach's modification of Giemsa's method. We have found this method very satisfactory for demonstrating bacteria in tissues. These sections were controlled by staining similar sections by Gram's method, and no gram-positive organisms were found.

COMMENT

An attempt to explain the apparent localization of microorganisms in the skin in cases of septicemia would be purely a speculative one, but would have to do probably with such factors as an extensive capillary bed, slower circulation, and the provision by the skin of a temperature more optimal for the growth of microorganisms. The fact that they are more easily demonstrated in the skin makes possible a method of early diagnosis, as has been emphasized by Netter and Salanier,[7] when they demonstrated meningococci in smears of the blood and tissue juices made from the purpuric areas. The cases presenting the skin lesions are often of a rapidly malignant character, and an earlier diagnosis, therefore, of a disease which responds to serotherapy may provide a way to more intelligent treatment and possible cure, whereas delayed diagnosis may result in the death of the patient.

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7. Netter, A., and Salanier, M.: Présence des méningocoques dans les éléments purpuriques de l'infection méningococcique, Compt. rend. Soc. de biol. **79**:755 (July 22) 1916.

CEREBROSPINAL MENINGITIS WITH PURULENT IRIDOCHOROIDITIS WITH RECOVERY*

REPORT OF A CASE

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The following case of cerebrospinal meningitis is presented as evidence of the striking effect of specific serum therapy not only on the disease itself, but also on one of the less common complications.

REPORT OF CASE

E. A., a girl, aged 18 months, whose father and mother are both healthy and born in the United States, was referred from the New York City Department of Health, Oct. 29, 1923, for admission to the Babies' Hospital and was under observation on the service of the late Dr. L. Emmett Holt for thirty-three days.

The patient was the youngest of four children; the others were apparently normal in every respect. Prior to the onset of the present illness the patient had been healthy, well nourished and well developed. She had been nursed for nine months, with supplementary feedings of cereals and fruit juice at six months. At nine months she was given a liberal balanced dietary.

Previous History.—Eight days prior to admission the child became acutely ill with fever, diarrhea and repeated vomiting. She became drowsy and, according to the mother's statement, slept almost continuously for three days. Four days after the onset the drowsiness entirely subsided and irritability and restlessness, both diurnal and nocturnal, persisted for the three ensuing days. On the sixth day the elevation of temperature, which had continued from the onset, rose to 105 F. and "black spots" made their appearance on the face and buttocks. Coincident with the appearance of the eruption it was noted that the child cried out whenever an attempt was made to change her position in bed and that she seemed to have great pain, especially in the neck, when her upper extremities were moved. On several occasions, on the sixth and seventh day after the onset, the mother noted that the eyes turned backward in the child's head and it was believed that she was having convulsions. Several of these "spells" occurred during the forty-eight hours prior to her admission to the Babies' Hospital, eight days after the onset of symptoms.

Physical Examination.—On examination the child was found to be moderately well nourished and well developed. She was semicomatose and could be aroused only with difficulty. A well marked purpuric eruption was present. This was most marked on the face, about the hips and shoulder girdle with a few spots irregularly distributed over the rest of the body. There was one spot the size of a dime on the hard palate. One large ecchymotic area 2 inches (5 cm.) in length, which subsequently became gangrenous, was present on the right side of the nose; there was also a smaller area on the buttocks, which also became gangrenous. Most of the petechial spots were the size of millet seeds, but there were many larger and a few of smaller size. The character of the eruption is well shown in the illustration.

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* Received for publication, April 9, 1924.
* From the Babies' Hospital.

The cornea of the left eye was markedly congested and the iris somewhat cloudy. There was no discharge. The right eye was normal. The tonsils were markedly enlarged and deeply injected. The heart and lungs were apparently normal. The abdomen was soft, and both spleen and liver could be palpated about 2 cm. below the costal margin. The anterior fontanel was about the size of a quarter and markedly bulging. The neck was rigid, the knee jerks were exaggerated and the Kernig and Brudzinski signs were both positive. The superficial lymph nodes were not enlarged.

Laboratory Examination.—Lumbar puncture was performed directly after the child's admission to the hospital, and 15 c.c. of cloudy cerebrospinal fluid was withdrawn under considerable pressure. The fluid contained 4,460 cells per cubic centimeter; the reaction for globulin was positive and the test for sugar negative. The smear revealed a great number of gram-negative diplococci, more of which were extracellular than intracellular. Blood examination showed 3,288,000 erythrocytes, 29,600 leukocytes of which 70 per cent. were

Petechial eruption of face, with gangrenous lesion on right side of nose. (Taken on day of admission to Hospital.)

polymorphonuclear neutrophils, 23 per cent. lymphocytes, 3 per cent. mononuclear leukocytes and the balance transitionals. The urine had a faint trace of albumin and a marked reaction for acetone; it was otherwise normal. Subsequent examinations of urine during the child's period of hospitalization were entirely normal.

It was decided to treat the disease with liberal amounts of antimeningococcus serum intravenously, combined with active spinal drainage and intraspinal serum administration. The table shows the amount and route of introduction. Both New York State and New York City Department of Health antimeningococcus serum was used.

Two days after admission, Dr. Martha Wollstein, the hospital pathologist, reported that the culture from the spinal fluid removed the preceding day showed a growth of Type 2 meningococci. (All subsequent laboratory examinations were done under the direction of Dr. Wollstein.) Blood taken at the same time for culture showed two days later many Type 2 meningococci. A routine Wassermann test was negative.

Clinical Notes.—The day following admission the general condition of the patient revealed no apparent change, but careful examination of the skin showed that no new petechial spots had appeared. On the third day the patient was less drowsy and could easily be aroused. From then on improvement was extremely rapid; by the sixth day the child became active, recognized objects and was obviously conscious of her environment. On the ninth day she recognized her parents, showed great interest in her food and responded to questions.

Slight rigidity of the neck was still present on the tenth day, but after a few days it entirely subsided. On this date the petechial eruption, with the exception of the two larger areas, one on the face and one on the buttocks, had entirely disappeared. The slough from these two areas exfoliated at the end of the second week. On the nineteenth day the right pre-auricular gland, which drained the gangrenous area on the nose, became swollen and tender. Coincident with the appearance of the adenitis there was a rise of temperature which persisted for several days.

Amount and Route of Injections of Antimeningococcus Serum

Date	Color	Pressure	Cells	Globulin	Sugar	Serum	Intraspinal, C.c.	Intravenous, C.c.	Spinal Fluid Chemistry per 100 C.c.
Oct. 29	Cloudy	++	4,460	+	—	State	20	25	Sugar 28 Chl. 725
Oct. 30	.....	....	.....	..	..	State	20	1 a.m. 25 9 a.m. 20	
Oct. 31	Cloudy	++	1,500	0	±	City	20	20	Sugar 64 Chl. 738
Nov. 1	Cloudy	++	560	+	±	City	20	20	Sugar 63 Chl. 713
Nov. 2	Turbid	+	222	+	—	City	12	..	Sugar 57 Chl. 713
Nov. 3	Clear	0	92	+	+	City	15	..	.....
Nov. 6	Clear	+	72	Trace	++	.....	..	..	.....
Nov. 8	Clear	+	8	—	+++	City	20	..	Sugar 71 Chl. 700
Nov. 9	Clear	+	1,540	+	++	City	20	..	Increase in cells due to meningeal reaction to serum
Nov. 15	Clear	+	9	+	+++	.....	..	..	Sugar 49 Chl. 713
Nov. 18	Clear	+	20	—	++	.....	..	..	.....

Complications.—On the day of admission it was noted that the iris of the left eye was markedly congested and the cornea cloudy. The second day the condition remained unchanged. The day following the corneal cloudiness had increased and the ciliary circulation seemed more congested. It seemed apparent that pus was forming in the anterior chamber. Dr. E. S. Thomson, who made an opthalmoscopic examination on the fourth day, and all subsequent examinations, made the following report: "There is no ciliary congestion and the conjunctiva is normal. There is marked lymph exudate in the anterior chamber from the iris and also through the pupil; the tension of the eye is slightly subnormal. There is evidently a purulent infection of the choroid extending to the iris and ciliary body. It is unusual in these cases to find iris exudation, and the appearance argues for a rapid virulent process. Panopthalmitis may possibly supervene. Diagnosis: purulent iridochoroiditis." Two days later it was noted by the attending staff that the gross appearance of the left eye had greatly improved. On the tenth day after admission it was observed that the iris was free from exudate but still somewhat discolored. There was a slight fundus reflex suggesting that most of the plastic exudate was in the anterior part of the vitreous chamber. On the fourteenth day after admission, examination revealed that the vitreous was steadily clearing up. The reflex

was brighter and a view of the fundus was almost possible. It was thought that prognosis was good as to vision. On the twenty-sixth hospital day the ciliary circulation was clear. The exudate back of the lens had absorbed, and there remained only a considerable amount of opacities and diffuse haziness of the vitreous. A clear view of the fundus could not be obtained but there was no appearance of a choroidal exudate. The vitreous was gradually clearing. Two weeks after the patient's discharge from the hospital it was observed that there was further improvement in the eye on the nasal side. In the ciliary region there was still some exudation. The tension of the eye was normal.

Six weeks after discharge Dr. Thomson found that improvement was progressive; that the mass of exudation in the ciliary region seemed to have almost entirely disappeared and only diffuse opacities were present in the vitreous, which were gradually lessening. A satisfactory view of the fundus was not obtainable.

The day following admission, both ear drums were incised and pus obtained from both. Gram-negative diplococci were demonstrated in smear and culture. Ten days after admission a characteristic serum rash appeared. The child's general condition rapidly improved, she sat up in bed about the sixteenth day and except for general muscular weakness seemed entirely well. One month after admission she was discharged as cured. The scar due to the gangrenous area on the right side of the face was plainly visible two weeks after discharge. Two months later the child was examined at the hospital; she walked about alone, played with toys and seemed to be normal in every respect.

COMMENT

Herrick [1] noted that of his 270 cases of epidemic cerebrospinal meningitis, only 11 had panophthalmitis; the infection reaching the eye through the ciliary vessels and not the optic nerve sheaths. He further observed that the cornea becomes cloudy and the iris congested. Shortly afterward the media becomes opaque and within a few hours sight is lost. In most cases enucleation had been performed, with resulting great improvement in the general condition of the patient. On microscopic examination the enucleated eyes showed meningococci in astonishingly large numbers. In a great number of cases mild congestive optic neuritis and retinitis were present in some degree and were a valuable sign toward early diagnosis.

In no case on record available to us has there been any report of complete restoration of vision after such an extensive invasion of the eye by the meningococcus. When the process has advanced to the stage of clouding of the transparent media, total destruction of the globe usually results. In a few cases the process has been arrested, but complete absorption of all products of inflammation has, we believe, never before been placed on record. The favorable result in the case herein reported is attributed by us to the vigorous treatment with antimeningococcic serum given by the intravenous as well as by the intraspinous route.

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1. Herrick, W. W.: Early Diagnosis and Intravenous Serum Treatment of Epidemic Cerebrospinal Meningitis, J. A. M. A. **71**:612-616 (Aug. 24) 1918.

Cases of this character illustrate the important factor of invasion of the blood stream in meningococcic infection; a point brought out so clearly by Herrick and his co-workers in the study of the initial stages of the disease made during the epidemic in the army camps in 1917 and 1918. They further illustrate the value of the intravenous administration of antimeningococcic serum in addition to the intraspinous method. These two points in our opinion, require vigorous emphasis. It is the practice at the Babies' Hospital to administer serum both intravenously, as well as intraspinously, in all cases of meningoccic infection.

PYLORIC HYPERTROPHY IN HYPERTROPHIC PYLORIC STENOSIS *

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Investigators have disagreed widely as to the relative size of the muscularis of the pylorus in the fetus, in the full term infant, and in the infant with pyloric stenosis. In their studies of the fetal pylorus Erich Moeller,[1] Jonnesco,[2] and A. Retzius[3] found the muscularis exceptionally prominent. In the fetus there is no pyloric valve as there is in the adult, and closure occurs by a contraction of the entire pyloric segment, which is known in the anatomic literature as the canalis pylori. They found the proximal boundary of the pyloric canal more or less indistinct, whereas the distal demarcation is usually rather sharp. In three premature infants Ibrahim[4] reported the muscularis of the pyloric canal thicker than that of the full term normal infant. Wachenheim[5] came to the conclusion that the diameter of the fetal pyloric musculature is often greater than that of infants a few weeks old. This he believes to furnish an "anlage" for subsequent hypertrophy. According to this author, a persistence of this transient, normal condition gives a primary anatomic basis for the origin of stenosis. If a disproportion in the antagonistic circular and longitudinal muscle layer exists, the one will develop more rapidly than the other and hypertrophy with its secondary symptoms of obstruction results. Hertz[6] failed to find the muscularis of the pyloric canal thicker in the fetus and prematurely born infant than in the full term infant. He considers a diameter of 448 to 672 microns, values which Fröhlich[7] found for prematurely born infants,

* Received for publication, March 1, 1924.

* From the Department of Pathology, University of Chicago, and the Sprague Memorial Institute Laboratory of the Children's Memorial Hospital, Chicago.

1. Mueller, E.: Beiträge zur Anatomie des menschlichen Foetus, K. Svenska Vetenskaps, Ak. Handl. **29**: Vol. 2, 1897.

2. Jonnesco: Traité d'anatomie humaine, Poierier **45,** 1895.

3. Retzius, A.: Bemerkunken über das Antrum pylori beim Menschen, Müller's Arch. **1**:74, 1857.

4. Ibrahim, J.: Die angeborene Pylorusstenose im Säuglingsalter, Berlin, 1905; Die Pylorusstenose der Säuglinge, Ergebn. d. inn. Med. u. Kinderh. **1**:208, 1908.

5. Wachenheim, F. L.: Pyloric Hypertrophy in New-Born Infants, Am. J. Dis. Child. **10**:87 (Aug.) 1915.

6. Hertz, P.: Ueber die angeborne Pylorusstenose des Säuglings., Jahrb. f. Kinderh. **72**:32, 131, 1916.

7. Fröhlich, T.: Om Pylorusstenose hos spaedbarn, Norsk. Mag. f. Laegevidensk. **4**:1046, 1906.

equal to that for full term infants. Cunningham,[8] on the contrary, claimed that this part is less developed during the last months of fetal life than in the new-born.

That the state of contraction of the normal stomach influences the thickness of its various parts is emphasized by Pfaundler.[9] He distinguishes a contracted or "systolic," from a relaxed or "diastolic" stomach and he maintains that a water pressure of 30 cm. will change the systolic into the diastolic type. He was probably the first to demonstrate the similarity in anatomy of the normal, systolic stomach and the hypertrophic pyloric stenosis stomach. Both have a thick muscle layer, deep rosette like folds of mucosa, and absence of a pyloric valve. The distal end of both is irregular, i. e., it is often longer at the greater than at the smaller curvature. This irregularity is caused by the diagonal course of the sulcus pyloricus. In both, the muscularis of the pyloric canal increases distally, reaching its greatest thickness at the end of the greater curvature. The chief difference between the normal systolic pylorus and the stenosed pylorus is an increase in muscularis of the latter. Pfaundler found the greatest thickness of the normal systolic pylorus to be 3 mm., that of the hypertrophic stenosis cases to range from 3.5 to 5 mm. Very recently, Wernstedt[10] has again asserted that to study the true thickness of the muscularis one must establish the "ground form" before the specimen is fixed and sectioned. He accomplishes this by exerting a water pressure of 30 cm., or more, on the immersed stomach. If the mucosal folds are thus obliterated there is no hypertrophy. Employing this preliminary technic he found the normal pyloric muscularis to average only 1 mm. in thickness; that of the hypertrophic pyloric stenosis cases to vary from 1.5 to 2.5 mm.

Most investigators report the hypertrophy to involve the circular muscle layer; Finkelstein,[11] however, reported a case in which the longitudinal layer was hypertrophied, and he and Gran state that all the layers of the pyloric wall may be hypertrophied. Bernheim,[12] Peden,[13] Wernstedt,[10] and Ibrahim,[4] who have made microscopic studies of the

8. Cunningham, J.: The Varying Form of the Stomach in Man and the Anthropoid Ape, Tr. Roy. Soc. Edin. **45**:1, 1906.

9. Pfaundler, M.: (Pylorusstenose im Säuglingsalter), Pfaundler-Schlossmann, Handbuch der Kinderkrankheiten **2**:181, 1906; Beiträge zur Frage der Pylorusstenose im Säuglingsalter, Jahrb. f. Kinderh. **70**:253, 1909.

10. Wernstedt, W.: Beiträge zum Studium des Säuglings-Pylorospasmus, Jahrb. f. Kinderh. **55**:674, 1907; Ungelöste Probleme in der Pylorusstenosefrage, Monatschr. f. Kinderh. **25**:676 (March) 1923.

11. Finkelstein, H.: Ueber angeborene Pylorusstenose im Säuglingsalter, Jahrb. f. Kinderh. **43**:105, 1896; Berl. klin. Wchnschr. **43**, 1897.

12. Bernheim: Ueber Pylorusstenose im Säuglingsalter, Cor.-Bl. f. schweiz. Aertze **8**:257, 1904.

13. Peden, W.: Case of Congenital Stenosis of the Pylorus in an Infant, Tr. Glasgow Path. & Clin. Soc. **3**:193, 1892.

muscle bundles, cells and nuclei, all agree that the individual muscle cells and nuclei are larger in the hypertrophic pylorus cases than in normal, contracted stomachs. They interpret their findings as proof of a true hypertrophy of the individual muscle cells.

The extensive, accurate measurements of the muscularis of the pylorus by Hertz [6] deserve consideration. He found, for example, that the diameter of the muscularis in two normal infants was 2,380 and 2,550 microns respectively; in some of his seventy newborn and prematurely born infants it reached 2,550 and 2,770 microns; and in his six pyloric stenosis cases it varied from 2,720 to 5,100 microns. These measurements show that in some of the normal and prematurely born infants the diameter of the pyloric muscularis approximated the lowest values found in the hypertrophic stenosis cases.

In spite of the fact that macroscopic inspection of the pylorus, accurate measurements of the diameter of the muscularis, and cytologic study of the muscle cells and their nuclei tend to show the existence of hypertrophy in stenosis cases, a number of investigators [14] question the existence of an actual increase in the muscle layer. Thus Pfaundler [9] takes the stand that the pylorus in these cases is merely in a state of spasm, and Haas [15] believes "hypertrophic pyloric stenosis to be an advanced degree of pylorospasm." Mohr [16] questions the validity of microscopic measurements in determining pyloric hypertrophy. The marked variations given by Hertz [4] and others for the thickness of the pyloric muscularis not only of stenosis cases but also of the normal, systolic pylorus make him ask: "When is a pylorus hypertrophied?" He reiterates that a micron is a very small unit of measure, that it is difficult to section specimens in exactly the same plane, and that it is almost impossible to obtain two stomachs in the same stage of contraction. Pfaundler [9] raises the objection that most of the microscopic studies have been based on material hardened in formaldehyd, and Aschoff [17] has demonstrated the marked influence of fixing fluids on the contraction of the pylorus.

The existence of the objections noted in the preceding paragraph, and the more or less conflicting data as furnished by accurate measurements, suggested to the author another line of attack in an attempt to prove definitely the existence of hypertrophy in pyloric stenosis. For this purpose wax models were prepared of the muscularis of two pylori

14. Sauer, L. W.: Hypertrophic Pyloric Stenosis—A Monographic Review, Arch. Pediat. **41**:145 (March) 1924.

15. Haas, S. V.: Congenital Hypertrophic Pyloric Stenosis and Its Treatment by Atropin, J. A. M. A. **79**:1314 (Oct. 14) 1922.

16. Mohr, M.: Ein Beitrag zur Frage des reinen Pylorospasmus, Ztschr. f. Kinderh. **29**:111 (June) 1921.

17. Aschoff, L.: Engpass des Magens, Jena, 1918.

of infants of the same sex, approximately the same birth weight, and practically the same age at the time of death. The control pylorus was that of an infant who died of peritonitis resulting from a ruptured appendix, the other one that of an infant who died of bronchopneumonia complicating a hypertrophic pyloric stenosis.

REPORT OF CASES

CASE 1 (Control).—F. P., a boy, was born at full term, September, 1922. The birth was normal, the weight was 3,050 gm. The child was nursed until the fatal illness which began five days before death. The weight was 2,820 gm. at the time of death, which occurred on the fifty-fifth day. Pathologic diagnosis: Appendicitis and peritonitis.

CASE 2.—(Hypertrophic pyloric stenosis).—J. W., a twin boy, was born September, 1922. The birth was normal, the weight was 2,800 gm. The child was nursed until a few days before death. The weight was 2,500 gm. at the time of death, which occurred on the fifty-second day. Clinical and pathologic diagnosis: Hypertrophic pyloric stenosis and bronchopneumonia.

The specimens taken from these cases, were selected from a collection of thirty infants' stomachs, and fixed in the following manner shortly after death. After the duodenum was tied off a few centimeters below the pylorus, the esophagus was cut a few centimeters above the cardia and the stomach contents expressed through the cardia. The stomachs were rinsed with Müller's fluid, and then were filled to capacity with the fluid; finally the esophagus was securely tied and the specimens were immersed in the fixing fluid.

After a few weeks the two specimens were removed from the fixing fluid, the pylorus was dissected from the rest of the stomach, and the two pylori were passed simultaneously through the various dilutions of alcohol, ether, and celloidin. They were then embedded in the same block of celloidin. The serial, longitudinal sections of 50 microns thickness were then stained with alum-hematoxylon and counterstained with acid fuchsin according to the Van Gieson method. There were 163 control sections and 238 sections from the stenosed pylorus. Tracings of the outline of the muscularis of each section were prepared with the aid of an Edinger projection apparatus at a magnification of twenty diameters. Finally, serial wax plates of 2 mm. thickness were prepared of every other section according to Born's method, and the muscularis of the pylorus was then reconstructed from the serial wax plates.

A glance at Figure 1 shows a distinct difference in the size of the two stomachs. The stenosis specimen is 5 cm. longer, 4 cm. wider at the cardia, and its capacity exceeds by 17 c.c. that of the control. It differs appreciably from the specimens examined by Hertz [6] and Wernsted [10] (Table of Data). Hertz found the size in most of the cases he examined to vary little from the normal, only three being distinctly

enlarged; the largest he studied had the following dimensions: greater curvature 31 cm., smaller curvature 7 cm., length 12.5 cm. and width 7 cm. Wernstedt found enlargement in none of the seventeen cases that he studied. Ibrahim,[4] Feer,[18] Pfaundler [9] and Adler [19] frequently found dilatation present. Hertz,[6] Hirschsprung,[20] Ibrahim [4] and Wernstedt [10] often found the muscularis of the entire stomach thicker than normal. This increases in diameter, as pointer out by Wernstedt, may be influenced by the state of contraction.

Inspection showed the cylindric, pyloric canal present in both specimens. The incisura angularis separated the pylorus proximally from the vestibulum (antrum), and the sulcus pyloricus marked it off sharply

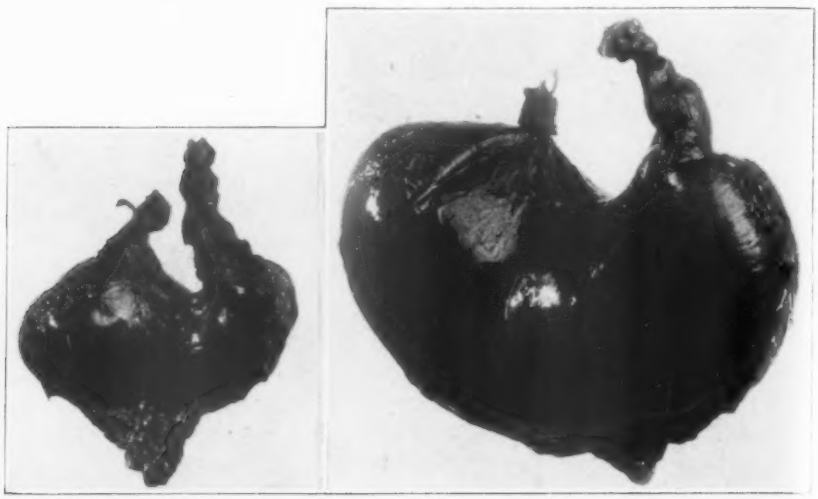

Fig. 1.—Dorsal view of the two stomachs; *a*, control case, *b*, stenosis case.

from the duodenum. Palpation aided in the determination of the distal boundary of both pylori, and inspection of the gastric mucosa was of value in locating the exact transition from vestibulum to pyloric canal, especially in the stenosis specimen, for the difference in width was greater and the gastric mucosal folds were absent (Fig. 2). The length measurement of the pyloric canal was made along a line equidistant from the smaller and the greater curvature. This is, in a sense, the average length, as the canal was found a few millimeters shorter at the smaller curvature, because of the diagonal, downward course of the

18. Feer, E.: Verh. Ges. Kinderheilk., Stuttgart, 1906.

19. Adler, M.: Ein Fall von angeborener funktioneller Pylorushyperplasie, Jahrb. f. Kinderh. **17**:197, 1908.

20. Hirschsprung, H.: Fälle angeborener Pylorusstenose. Jahrb. f. Kinderh. **28**:61, 1888.

sulcus pyloricus from the smaller to the greater curvature. The cartilage-like specimen is somewhat curved, with its convexity along the great curvature. The control specimen measured 18 mm., the stenosed pylorus 23 mm. Hertz found the pyloric canal of the newborn to measure from 12 to 18 mm., that of stenosis cases to vary from 15 to 30 mm. with an average of 20 mm.

Microscopic examination of the stained, longitudinal sections of both pylori (Fig. 3) showed the presence of four layers: (1) Tunica serosa (visceral peritoneum); (2) tunica muscularis, consisting of: (*a*) an outer, longitudinal layer (stratum longitudinale); (*b*) an inner, circular (stratum circulare); (3) tela submucosa, an appreciable layer

Fig. 2.—Control and stenosis pylori, showing *a*, incisura angularis; *b*, sulcus pyloricus; *c*, vestibulum (antrum) pylori.

of loose connective tissue, elastic fibers, fat, and blood vessels, and (4) tela mucosa, the thin, innermost layer of mucous membrane.

The striking difference in the two specimens was the marked increase in the stratum circulare of the tunica muscularis in the stenosis specimen (Fig. 3). The greatest diameter of the tunica muscularis measured 3,850 microns; that of the control specimen was 2,500 microns. Hertz found the diameter in his stenosis cases to average between 3,000 and 4,000 microns, although in one instance it measured only 2,750 microns, a value equal to that found by Wernstedt [10] for a normal,

21. Simon, J.: Anatomischer Magenbefund eines Säuglings fünf Monate nach Heilung von Pylorusstenose, Jahrb. f. Kinderh. **63**:331, 1911.

4 months old infant. Ibrahim [4] and Simon [21] obtained no measurements above 2,000 microns for the contracted pylorus of the normal new-born.

The mucosa was not found increased in the stenosis case. It was characteristically raised into several more or less longitudinal folds which, so far as could be determined, extended throughout the pyloric canal in both specimens. Although the mucosa and the submucosa have been reported thickened in several cases of pyloric stenosis, all of Wernstedt's sixteen cases failed to show such a variation. The results of his investigations are supported by the fact that in Figure 3 *B* the diameter of the mucosa is certainly not greater than that in the control,

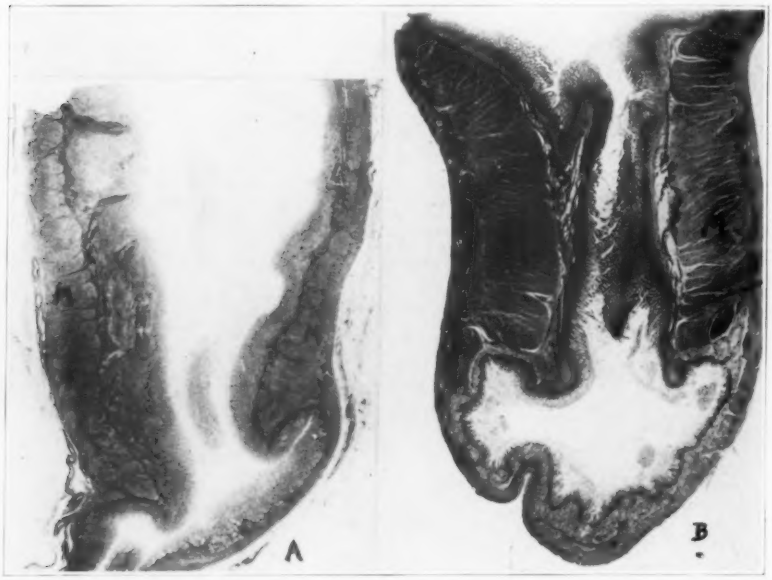

Fig. 3.—Longitudinal section of the pyloric canal; *a*, control case, *b*, stenosis case. Marked increase in muscle layer (u) in *b*.

Figure 3 *A*. Bendix [22] holds that the mucosa may contribute appreciably to the obstruction of the lumen by producing folds which effect a valve-like closure. The diameter of the pyloric lumen cannot be accurately measured because the mucosal folds produce more or less obliteration. In fatal cases of hypertrophic pyloric stenosis, the lumen has been found to vary from the diameter of an average sized lead pencil to that of the finest sound. Hertz attempted to measure the lumen in two of his fatal stenosis cases by sectioning the pylorus longitudinally and then

22. Bendix, B.: Pylorospasmus und Pylorusstenose im Säuglingsalter, Med. Klin. **5**:1813, 1909.

stretching the specimen on a flat surface. The distance between the cut surfaces measured 18 mm. in the one instance, and 22 mm. in the other. These measurements are almost the same as those made by

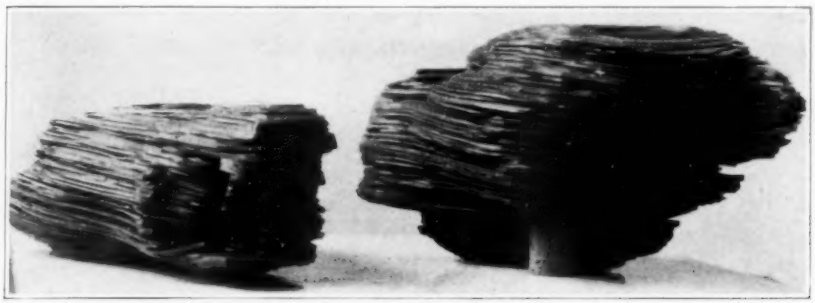

Fig. 4.—Wax models of the muscular layer of the pyloric canal; *a*, normal pylorus, *b*, stenosis case.

Fig. 5.—Finished models, showing contrast in size, also diameter of lumen, and thickness of the muscularis.

Fig. 6.—Lateral view of models, showing the shape of the hypertrophied, pyloric muscularis at X.

Pfaundler on thirteen infants, that died during the first month of life. A reduction of the lumen is, therefore, not due to a decrease in the mucosal surface.

That an actual increase in muscularis occurs in pyloric stenosis is emphasized by a comparison of the two wax models. This evidence is more conclusive than microscopic measurements of the diameter of the muscularis, for we know that the spastic element in stenosis makes the muscularis of the pylorus contract, and its cartilage-like consistency is more likely due to the spastic contracture than to an increase in the

A Comparative Tabulation of Data

	F. P. (Normal)	J. W. (Hypertrophic Pyloric Stenosis)
STOMACH		
State	Systole	Diastole
Capacity	37 c.c.	54 c.c.
Length	8 c.m.	13 cm.
Width at cardia	4 c.m.	8 cm.
Mucosa	Deep folds	No folds
PYLORUS		
State	Patent	Not patent
Lumen	Rosettelike folds	Deep, rosettelike folds
Consistency	firm	Cartilaginous
Length	18 m.m.	23 mm.
Circumference	38 m.m.	47 mm.
Number of sections	163	238
MODEL		
Weight	3,000 gm.	6,050 gm.
Length	30 cm.	35 cm.
Height	14 cm.	22 cm.
Median diameter	22 cm.	28 cm.
Median circumference	57 cm.	76 cm.
Thickness at antrum	2 cm.	4 cm.
Thickness at duodenum	2.5 cm.	9 cm.
Plane of lumen	Horizontal throughout	Horizontal, vertical, horizontal
Diameter of lumen at antrum	18 cm.	18 cm.
Median diameter of lumen	12 cm.	8 cm.
Diameter of lumen at duodenum	4 cm. x 7 cm.	9 cm. x 12 cm.
Form of lumen	Funnelshaped	X-shaped

muscle layer. Figure 4 illustrates the models of the muscularis *A* of the normal pyloric canal; *B*, of the infant with pyloric stenosis. Model *A* weighs 3,000 gm. and model *B* weighs 6,050 gm. This vast difference in bulk is conclusive evidence, as the infant with the stenosis weighed 150 gm. less at birth, never regained his birth weight, weighed approximately 300 gm. less at the time of death, and was three days younger than the control. The finished models (Fig. 5.) illustrate not only the marked contrast in size, but also the diameter of the lumen and the thickness of the muscularis of the two specimens.

A comparative study of the two models shows not only that they differ in mass, but that the form and plane of the lumina are not alike.

The so-called lumen or passageway in the control model resembles somewhat a funnel with the smaller aperture at the distal or duodenal end, whereas in the stenosis model it is narrowest near the middle, thus resembling somewhat the letter X. These differences can also be seen in the longitudinal sections (Fig. 3), but as this difference involves mainly the muscularis, it is more pronounced in the models. The measurements of the lumen at different levels (as recorded in the Comparative Tabulation of Data), verify this point. The other difference which deserves mention is the plane of the so-called lumen. In the control model it remains horizontal from one end of the pyloric canal to the other; in the stenosis model, on the other hand, it is first horizontal, then there is a rather abrupt vertical drop near the middle of the model, and from there on it continues horizontal to the distal end. Further investigation of these two striking differences may lead to a solution of the etiology of hypertrophic pyloric stenosis.

SUMMARY

1. Evidence offered as proof of hypertrophy of the tunica muscularis of the canalis pylori in infants with pyloric stenosis has been challenged by various investigators.

2. Wax models of the muscularis were prepared from serial, longitudinal sections of two pylori from male infants of approximately the same age and weight, *A* a normal pylorus and *B*, a typical stenosis of the pylorus.

3. The lumen of the control model is somewhat funnelshaped; that of the stenosis model resembles somewhat the letter X.

4. The plane of the lumen of the control model is horizontal; that of the stenosis model is horizontal, vertical, horizontal.

5. The wax model of the normal muscularis weighed 3,000 gm., that of the stenosis weighed 6,050 gm., a conclusive demonstration that a marked hypertrophy of the muscularis exists.

PROGRESS IN PEDIATRICS. REVIEW OF THE LITERATURE FOR 1923 ON INFANT FEEDING*

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BREAST FEEDING

Kuttner and Ratner [1] have undertaken to investigate the work of Theobald Smith to determine the essential nature of colostrum feeding in calves. These investigators were interested in its bearing on infant feeding. Smith showed that colostrum is not alone the chief means for transferring antibodies to the new-born calf. Colostrum also seems to have the important function of protecting the new-born animal against a general invasion by organisms of the *bacillus coli* group. With this in mind, Kuttner and Ratner sought for evidence of histologic differences in the placentas of various species. They used in their researches toxin, antitoxin and the Schick test as a means of control. There were some investigators who claimed that the placenta was impermeable to antibodies and that only through the milk was immunity transferred. In approaching this problem in human beings it was divided into two lines of study: (1) to determine the manner in which antibodies are conveyed from mother to child; comparing the antibody contents of colostrum with that of the blood of the mother, the cord blood, and the blood of the infant after the ingestion of colostrum, and (2) by clinical study of any reliable records of noncolostrum fed infants.

The result of their inquiry produced the following information. The human placenta was found, in accordance with the results of previous workers, to be penetratable to diphtheria antitoxin. Human colostrum occasionally contains small amounts of antitoxin, but this amount is always less per volume than that found in the blood serum of the mother or in the cord blood. No antitoxin was shown to exist in the colostrum. They concluded that colostrum feeding in human beings has no significance as to transferring immunity. These findings do not agree with those of Theobald Smith.

During the last year encouragement to the young mother to nurse her child has received a great deal of attention by the profession. Donnelly,[2] in an extensive article on breast feeding, discusses the

* Received for publication, April 23, 1924.

1. Kuttner, Ann, and Ratner, Bret: The Importance of Colostrum to the New-Born Infant, Am. J. Dis. Child. **25**:413 (June) 1923.

2. Donnelly: Methods of Sustaining and Regulating the Supply of Breast Milk, Long Island M. J. **17**:13 (Jan.) 1923.

methods of sustaining and regulating the supply of breast milk. He believes that two fundamentals are necessary: (1) regular stimulus to the breasts by the suckling infant, and (2) complete emptying of the breasts at each feeding. The quantity of breast milk may be increased in four practical ways: (1) by nursing both breasts at each feeding, (2) by increasing the intake of fluids, (3) by proper exercise, short of fatigue, and (4) by securing sufficient rest at night. The solids in the milk are increased by shortening the intervals of nursing and are diminished by lengthening these intervals. He believes that the vitamin content is of great importance. He instructs the mother to take large quantities of foods that are rich in vitamins. Both Richardson [3] and Watton [4] urge that every new-born infant delivered in a hospital ward be immediately placed on the children's service, so that the pediatrist can look after the supply of mother's milk and conserve it for as long as possible. Proper campaigns should be launched to instruct mothers in the necessity of breast feeding for some months; together with the technic of breast feeding. Emphasis should be placed on the manual emptying of the breasts. The manipulation is quite similar to that employed in ordinary milking, except that approximation of the thumb and finger is substituted for the whole hand. The plea for supervision by the physician, who is interested in child welfare, is important at an early age.

Jewesbury [5] has been able to continue breast feeding in at least 95 per cent. of the cases, if not alone, at least partly. The only cases where breast feeding should not be advised is when the mother is suffering from active pulmonary tuberculosis, or any disease in which there might be rapid progressive wasting, or when she again becomes pregnant. In the presence of failing milk he puts the baby regularly to both breasts, usually at three hour intervals. In Schoedel's [6] experience the weak infant who is doing well will drink at the end of the second week from 130 to 175 gm., at the end of the third week from 140 to 190 gm., at the end of the fourth week from 145 to 210 gm., and from the sixth to eighth weeks from 150 to 250 gm., per kilogram of body weight. For a weak infant to thrive well on exclusive feeding with pumped milk it is often desirable to give him somewhat larger quantities than this. In his clinic, the children make good progress when they drink at the

3. Richardson: A Breast Feeding Campaign, Long Island M. J. **17**:9 (Jan.) 1923.

4. Watton: Supervising Breast Feeding, Long Island M. J. **17**:14 (Jan.) 1923.

5. Jewesbury, R. C.: Some Notes on Breast Feeding, Arch. Pediat. **40**:101 (Feb.) 1923.

6. Schoedel, J.: The Amount and the Number of Feedings on which Weak Infants Thrive when Fed on Breast Milk, Jahrb. f. Kinderh. **102**:227 (June) 1923.

end of the first week from 95 to 110 gm., and at the end of the second week from 140 to 240 gm. His figures show the need for greater amounts of food for weak infants. He says that it should always be remembered that the sucking power of the weak infant is soon exhausted, and hence if put to the breast more frequently it will obtain greater amounts of food. He prefers giving the breast feeding eight times in twenty-four hours for the premature baby and decreases these to seven or six as the child develops. The advantage of more frequent stimulus to the breasts increases their function, which is endangered if the child drinks with very little force at long intervals. Weak or premature infants are usually given from eight to ten feedings.

In discussing the methods for promoting the supply of breast milk Smyly [7] believes that the rules are so simple that the poorest mother can carry them out in the poorest home. This work in China has been directed to institute a simple, healthy, well regulated life amid hygienic surroundings. The mother and baby are kept in the open air and sunlight as much as possible. The mother should take morning exercises before an open window every day; this should never be carried to the point of fatigue. Alternate hot and cold sponging of the breasts should be performed morning and evening. A definite period should be arranged each day when the baby's bulky clothing can be removed, allowing the free use of its limbs. The results in two cases are reported: (1) the milk supply was reestablished after nursing had been entirely discontinued for a week, (2) a patient with low mentality and poor physique was able to nurse her child satisfactorily after following a definite program.

Birk [8] has made a study of the changes in the breast milk during lactation. He employed in his clinic the same woman as a wet nurse for three years. This gave him an opportunity to determine whether the content of nutritive material and salts still averaged the minimum physiologic requirement. He found that fluctuation in the amount of protein was insignificant. The average value of the milk fat was 5.4 per cent., lactose varied from 7.2 to 7.6 per cent. He believes that breast milk, after three years of lactation, still contains a sufficient amount of nutritive material and salts. Apparently the figures given by other authors for breast milk have habitually been made at the beginning and at the height of lactation.

The question of whether the infant may thrive better on cooked breast milk than on raw breast milk has been studied in great detail by

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7. Smyly, E.: Deficiency of Breast Milk in Nursing Mothers: Treatment, China M. J. **37**:834 (Oct.) 1923.

8. Birk, W.: Changes in Breast Milk During Lactation, Monatschr. f. Kinderh. **25**:30 (March 25) 1923.

Martin[9] and Wülffing.[10] Fifty new-born and older infants were fed on boiled breast milk. The breasts were milked alternately at the feeding time. The milk was caught in sterile vessels and put into bottles which were sterilized by boiling vigorously. As colostrum coagulates on boiling, the new-born infants were given boiled milk from other mothers until their own mothers' milk became free from flocculation. There was no noticeable thickening of the milk on boiling. The weight curves of these children were the same as those of breast fed children, or even a little more favorable. The quantity of the breast milk was increased by careful milking. This, he believes, shatters the theory that it is stimulated by sucking. Boiled breast milk is as good a food as unboiled for the normal infant. At any rate cooked breast milk does not have any unfavorable effect.

In discussing the use of complementary breast feeding Gerdine[11] feels that this method has grown popular throughout the country and is of great value. It consists of putting the infant to the breast at regular feeding times and encouraging it to nurse as completely as possible. The breasts are then emptied by the manual method, as he feels that this is vastly superior to any pump. In this way it is recognized that practically every mother can nurse her baby.

Woodbury,[12] in a study on the relationship between breast and artificial feeding as a cause of infant mortality, obtained data from eight American cities, through the Children's Bureau in Washington. The information included a record of the feedings received in each month of life for the first year. An analysis on the influence of feeding was made from data on 22,422 live born infants. The kinds of feeding were classified into three groups: (1) exclusively breast, (2) exclusively artificial, and (3) partly breast and partly artificial. The analysis showed that artificial feeding, as actually practiced in typical American city populations, is associated with a mortality of between three and four times as high as that among breast fed infants. This excess is not to be explained by the fact that the artificially fed infants form a much larger group than the others; it appears that this mortality goes through all nationalities and all earning groups, though with slight variations depending possibly on the particular conditions prevailing in each group.

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9. Martin, E.: Boiled Breast Milk, Klin.-therap. Wchnschr. **2**:228 (Feb. 12) 1923.

10. Wülffing, E.: Feeding with Cooked Breast Milk, Ztschr. f. Kinderh. **35**: 56 (Feb. 14) 1923.

11. Gerdine, L.: Complemental Breast Feeding, J. M. A. Georgia **12**:4 (Jan.) 1923.

12. Woodbury, R. M.: The Relation between Breast and Artificial Feeding and Infant Mortality, Am. J. Hygiene **2**:668 (Nov.) 1922.

ARTIFICIAL FEEDING

During the last year very little has been added to our knowledge of artificial feeding. Articles by Giffen,[13] Cowie,[14] Hoobler,[15] Mucklow,[16] Mixsell,[17] and Schmalfuss [18] have been published on this subject. They all agree that the normal infant requires 45 calories per pound of body weight, to maintain and gain weight. Whole milk mixtures with simple dilutions of plain or barley water, with the addition of sugars, seem to be most popular. Probably the greatest addition to infant feeding during the last year has been the more extensive use of lactic acid milk and corn syrup. Almost every type and combination of feeding has been tried.

In a very complete and exhaustive survey of the whole subject on the pathology and therapy of nutritional disorders in artificially fed infants Finkelstein [19] divides the cases of diarrheas into the following groups: (1) Fermentation diarrheas. These are brought about by the irritative effects of volatile fatty acids, which have a stimulating effect on the peristalsis. As compared with fermentation, putrefaction is of minor significance as an agent producing diarrheas in infants. It has been shown by clinical observations, as well as by experiments, that in fermentation we deal with a carbohydrate fermentation. The crystalline sugars, lactose above all, are particularly related to diarrheas. There is a remarkable difference between the crystalline and the colloidal carbohydrates, including flour. As regards fermentability in the intestine, polysaccharids are much more resistant to bacteria, but show a depressing influence on the velocity and intensity of sugar fermentation. He believes that fat turns the balance, increasing diarrhea when there is a tendency to fermentation, whereas it favors the formation of solid stools in the opposite case. We should never forget that in the intestinal chemistry the influence of metabolic dynamics on nutrition has a decisive significance. The same food may, under varying condition, in different children or with the same child at different times, produce a

13. Giffen, S. D.: The Feeding of Infants Based on Recent Experience, Ohio State M. J. **18**:829 (Dec. 1) 1922.

14. Cowie, D. M.: A Simple, Practical Method for the Feeding of Infants, J. Michigan M. Soc. **22**:10 (Jan.) 1923.

15. Hoobler, B. R.: Feeding of Infants and Children, J. Mich. M. S. **22**:16 (Jan.) 1923.

16. Mucklow, A. E.: Infant Feeding, New York M. J. **117**:283 (March 7) 1923.

17. Mixsell, H. R.: Some Newer Methods of Infant Feeding, J. M. Soc. New Jersey **20**:122 (April) 1923.

18. Schmalfuss, M.: The Technic and Objects of Mixed Milk Feedings, Monatschr. f. Kinderh. **26**:387 (July) 1923.

19. Finkelstein: On the Pathology and Therapy of Nutritional Disorders of Artificially Fed Infants, Yearly Courses for Physicians, Berlin, **1**, No. 6, June 1923.

different effect. Protein is not only valued as a stimulant to fermentation, but as a buffer substance by promoting the secretion of intestinal juices. Protein, therefore, helps to neutralize acids, to accelerate the digestion and absorption. Crystalline sugars are not merely a substratum for fermentation, but show practically the identical action as in the test tube. Lactose seems to deserve the foremost rank as a substratum for enzymatic action. All things being equal, lactose has the most powerful purgative effect. It acts more intensely than the crystalline saccharose. He calls attention to the fact that the decisive factor in the origin of diarrheas is not to be found in abnormal fermentative processes. The production of abnormal acid reactions, in parts of the intestine very sensitive to acids, causes diarrheas. Therefore, an abnormal decomposition in the jejunum makes the diarrhea more intense the higher in the intestine the process of decomposition takes place; (2) Inanition diarrheas. These may occur if the food ingested is deficient in the one indispensable constituent. In infants, it is usually part of a terminal condition. In extreme atrophic children, very often during the last days of life without any particular reason, watery stools occur with an abrupt loss of weight, leading very soon to a fatal collapse. According to Finkelstein's experience this condition is relatively frequent in the first days of life, probably because of the fear of doing harm to the new-born infant by giving cow's milk in sufficient amount. The most frequent cause is possibly an insufficient ingestion of carbohydrates. At times, even a slight deficiency in the requirements may be of grave consequence. The severe cases of atrophy are usually due to a hydrolabile disposition, as they often show a tendency to diarrhea because of nutritive or carbohydrate deficiency; (3) Irritation diarrheas. In the vast majority of cases irritation diarrhea is brought about by an infection of the colon and ileum. These conditions resemble the catarrh of the air passages. They develop in proportion to the amount of immunity and resistance that is present. In an organ weak in its power of resistance bacteria become pathogenic; while in a healthy organ they are almost harmless. These three groups of diarrhea cannot be sharply distinguished as they may be associated with one another in various ways. In each case we have to deal with secondary inflammation of the intestinal tract.

Horwitt,[20] following the program of the German school, believes that their figures are the normal requirements for healthy infants. One-tenth of the body weight in milk and one-hundredth of the body weight in sugar or carbohydrate. He objects to the name of caloric

20. Horwitt, S.: Theory and Practice of Artificial Infant Feeding, New York M. J. **117**:278 (March 7) 1923.

feeding, yet he works out his feeding program on the basis of 45 calories for each pound, and where there has been undernourishment he has given as many as from 50 to 60 calories.

BUTTER FLOUR MIXTURES

All over Europe where there has been a deficiency of milk, the Czerny-Kleinschmidt butter flour feeding has gained in popularity. Articles by Gismondi,[21] Lecoq,[22] Ernberg,[23] Lowenburg,[24] Hamburger,[25] Schwartz,[26] Kasteele,[27] Poulsen[28] and Becking[29] describe the usual method of preparing butter flour, as laid down by Czerny-Kleinschmidt in their article in 1918.

Lowenburg[24] feels that fat feeding is sometimes a great problem. To find a way of supplying this food in sufficient amount and in acceptable form for the digestive apparatus, he has tried to ascertain why it is possible to feed large amounts fat in the butter flour mixture, when the same amount of cream or top milk added to a formula could not be tolerated. There seems to be some advantage in the use of boiled butter over raw butter and cream. The presence of flour or starch seems to insure an even distribution of the fats. Hamburger[25] mentions the use of complementary flour feedings in healthy breast fed children and gives his results. For healthy artificially fed children he substitutes butter flour mixtures for some of the carbohydrate gruels, used to dilute the milk, without changing the rest of the diet. The whole amount of butter is 5 per cent., combined with 5 per cent. flour. In cases of atrophy and dyspepsia he uses one-half buttermilk with varying amounts of sugar, to which 50 gm. of butter-flour porridge is added. He has noted in the new-born and premature baby an intolerance to this form of fat. Poulsen[28] has presented a report on his experience with the use of butter-flour feeding in children under one year. He prepared his

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21. Gismondi: The Butter-Flour Food of Czerny and Kleinschmdit in the Feeding of Infants, Riv. di clin. pediat. **20**:577 (Oct.) 1922.

22. Lecoq: Lactated and Other Flours and Meals Employed for Artificial Feeding, Soc. Sc. d'hgy. aliment. **10**:552 (Nov.) 1922.

23. Ernberg: Experiences with a Butter-Flour Dietary, Acta pediat. **2**:149 (Dec. 16) 1922.

24. Lowenburg, H.: A Comparative Chemical and Clinical Study of Boiled Butter and Cream in Infant Feeding, New York M. J. **117**:295 (March 25) 1923.

25. Hamburger, R.: Further Experiments with Butter-Flour Feedings, Monatschr. f. Kinderh. **25**:254 (March 25) 1923.

26. Schwartz, A. B.: The Present Status of Infant Feeding with the Butter-Flour Mixture of Czerny-Kleinschmidt, Wisconsin M. J. **21**:539 (May) 1923.

27. Kasteele: Experiences with Butter-Flour Feedings in the Milk Station of the Fishing Hamlet of Schevenignen, Monatschr. f. Kinderh. **25**:314 (March 25) 1923.

28. Poulsen, V.: Experiences wtih the Czerny-Kleinschmidt Butter-Flour Feedings, Monatschr. f. Kinderh. **25**:539 (March 25) 1923.

29. Becking: Results with the Czerny-Kleinschmidt Butter-Flour Feeding at the Utrecht Pediatric Clinic. Nederl. Maandschr. v. Geneesk. **12**:1 (July) 1923.

mixture according to the original formula of Czerny-Kleinschmidt.[30] He gives as much as from 200 to 250 gm. per kilogram of body weight each day. Forty-three cases showed a weekly gain of from 200 to 300 gm. In no instance was the butter-flour diet continued for more than three months. The child could then be discharged from the hospital as it had become accustomed to a normal diet.

In studying the literature on the use of butter-flour feeding, one cannot help but feel that in America the necessity for the use of this preparation is not as urgent as in Germany. The average good milk in America contains 4 per cent. fat, while in Germany the milk contains between 1 and 2 per cent. The addition of butter then becomes a necessity.

ACIDIFIED MILK

Nuller[31] studied the buffer value of cow's milk, both full milk and whey, on the basis of accurate electrometric titration curves. Cow's milk proved to be especially well buffered at from p_H 6 to 4.5. The buffering action of whey albumin is extremely slight. Casein, on the other hand, has an extensive buffering action which causes the strong buffering of cow's milk in the p_H region of from 6 to 4. The different position of hydrogen-ion concentration on milk feeding and on meat diet is explained by the different buffering capacity of the gastric juice which is produced. The buffering of the milk predominates in the gastric needs of infants. Babbott[32] made 227 observations on the hydrogen-ion concentration of the gastric contents in forty-three infants, from 3 to 19 months of age. For the test meal a powdered milk preparation, dissolved in distilled water, was used because its composition remained constant and its concentration could be varied at will. The composition of this mixture was fat 12 per cent., lactose 46 per cent., protein 32 per cent., salt 7 per cent. and moisture 3 per cent. A formula of 8 parts powdered milk to 100 parts water was given because it corresponded approximately to skimmed milk. The hydrogen-ion concentration of this meal was determined colorimetrically as 6.2. The normal range of hydrogen-ion concentration of the gastric contents on the milk test meal at the end of one hour was from p_H 3 to 2.5 with 76 per cent. of the readings between p_H 3.9 and 4.6. Acid production and hydrogen-ion concentration increases with increase of age. The greater the gastric acidity the more rapidly the stomach empties. The secretory capacity of

30. Czerny-Kleinschmidt: Butter-Flour Feeding, Jahrb. f. Kinderh. 1918.

31. Nuller: The Buffer Capacity of Cow's Milk. Ztschr. f. Kinderh. **35**:285 (June 7) 1923.

32. Babbott, F. L.; Johnston, J. A.; Haskins, C. H., and Shohl, A. T.: Hydrogen-Ion Concentration of Gastric Contents of Infants, Am. J. Dis. Child. **26**:475 (Nov.) 1923.

infants is limited, so that when larger amounts of food are given they are no longer able to reach the optimal acidity.

Rosenbaum [33] has tried to determine whether the addition of hydrochloric acid causes more rapid emptying of the stomach. He finds that protein catabolism does not occur after mere addition of hydrochloric acid, up to the usual degree of acidity in preliminary peptic digestion. The excess of buffer value in cow's milk delays, in many cases, the gastric digestion in infancy by inhibiting the necessary acidity for the efficiency of gastric enzymes. To overcome this deficiency Faber [34] has suggested modifying cow's milk by addition of 0.4 per cent. hydrochloric acid. For this modification 20 c.c. of acid is added to each 100 c.c. of milk and the preparation called 25 per cent. hydrochloric acid milk. This milk, with its reaction of p_H 6, is slightly acid; it is ingested by infants without the slightest protest. The acid is taken up largely by the basic phosphates and corresponding chlorids. The increase in soluble calcium salts may be an additional advantage. No free acid is present in the milk, yet there is sufficient acidity to inhibit the growth of bacteria. In his experience the effect of this milk is to produce lime soap stools, like those from protein milk and to affect favorably the transition of fats, but it does not influence the digestion of sugars. Usually the transition from hydrochloric acid milk to an ordinary milk formula is easy. He does not believe that acidified milk should be given to infants during the first 6 weeks of life.

In studying the acidity of the gastric contents in infants, Marriott [35] examined forty-one normal infants under 1 year of age. Twenty-three infants were breast fed and seventeen received breast milk from the bottle. The average hydrogen-ion concentration of the gastric contents for the whole series was 3.75. In a group of sixteen infants, most of them extremely undernourished, including cases of infection (otitis media, pneumonia, dysentery), the gastric contents showed a hydrogen-ion concentration of from 4.4 to 4.8; the average being 4.74. In order to overcome the high buffer value of sweet cow's milk, which neutralizes a considerable portion of the acid of the gastric juice, the addition of one teaspoonful of lactic acid (U. S. P. from 75 to 85 per cent.) to each pint of milk is necessary. This is a sufficient amount to bring the acidity of the gastric contents approximately up to normal. Besides the important effect of increasing the acidity of the gastric contents it has an antiseptic action. It also stimulates the flow of bile,

33. Rosenbaum, E.: Gastric Secretion in Infants, Monatschr. f. Kinderh. **24**:487 (Jan.-Feb.) 1923.

34. Faber, H. K.: Hydrochloric Acid Milk in Infant Feeding, Am. J. Dis. Child. **26**:401 (Nov.) 1923.

35. Marriott, W. McK., and Davidson, L. T.: The Acidity of the Gastric Contents of Infants, Am. J. Dis. Child. **26**:542 (Dec.) 1923.

pancreatic juice, and intestinal secretion. Marriott [36] has combined the use of corn syrup with the lactic acid milk mixture with very gratifying results. Corn syrup is used in the same proportion as granulated sugar, 120 calories for each ounce. Dohnal [37] has examined the acidity of the gastric juice when from 50 to 100 gm. buttermilk were given after from one to three breast feedings. Forty-five minutes after the feeding the free and combined hydrochloric acids were estimated. Later, the actual amount of hydrochloric acid was determined by Barun's ash method. Tables show that there were daily and hourly variations in acidity valuation. After buttermilk there were high degrees of acidity, which were as large as the amounts of acid introduced into the stomach. In prematurely born children the favorable effect of buttermilk seems to be partly due to hydrochloric acid excretion. The result of his examinations showed that the acidity of the gastric juice in infants is variable, while the hydrochloric acid concentration is constant. The hydrochloric acid is increased by buttermilk. These facts indicate the advantage of giving buttermilk.

GOAT'S MILK

Brouwer,[38] quoting the work of Schemtema, noticed that many children fed on goat's milk suffered from severe anemia, which was frequently accompanied by great enlargement of the spleen and sometimes enlargement of the liver. He reports five patients, aged 9, 9, 12½, 8 years, and 5 months. Breast milk was either not given or given for too short a time. The goat's milk was diluted one-half with water, adding a moderate amount of flour and sugar. All the children had loss of appetite, vomiting, and diarrhea. Their skin was pale and slightly yellow, with changes in the bony structure similar to rickets, and they were underweight. The blood picture resembled that of von Jaksch's anemia. The leukocyte count was high, reaching 70,000; the percentage of polymorphonuclear leukocytes was small, with myelocytes always present. Czerny-Kleinschmidt thinks that one-sided feeding with milk is an etiologic factor in von Jaksch's anemia. In order to confirm the alimentary character of the anemia they treated the gastrointestinal disturbance with albumin milk and then buttermilk. The children recovered rapidly. This anemia did not occur when breast milk was given with goat's milk, but where goat's milk was given early when breast milk could no longer be had, the anemia occurred in every case. In thirty-nine cases of severe and moderately severe anemia, in children

36. Marriott, W. McK., and Davidson, L. T.: Acidified Whole Milk as a Routine Infant Food, J. A. M. A. **81**:2007 (Dec. 15) 1923.

37. Dohnal, E.: The Action of Buttermilk on Stomach Secretion, Monatschr. f. Kinderh. **27**:58 (Oct.) 1923.

38. Brouwer, E.: Goat's Milk Anemia and Goat's Milk Feedings, Jahrb. f. Kinderh. **102**:257 (July) 1923.

under 2 years of age, it was definitely stated that twenty had been fed with goat's milk; most of the children were less than 1 year of age. From 1914 to the end of 1920 goat's milk was the most frequent cause of anemia in children. Goat's milk anemia is a mild blood disease, but, in spite of this, deaths have occurred. In young infants breast milk naturally deserves the preference. After recovery from the gastro-intestinal disturbance, fruit juices, vegetables and cereals can be given. Frequent examination of the blood picture is necessary. In very severe cases blood transfusion should be tried.

THICK CEREAL FEEDING

In his three years' experience with the use of thick cereal feeding Mixsell [39] reports some splendid results. His indications for the use of this preparation are: (1) malnutrition (marasmus), (2) pylorospasm (mild or severe), (3) pyloric stenosis, (4) breast fed colic cases, (5) persistent vomiting (neuropathic), (6) idiosyncrasy for breast or cow's milk, (7) liquid food intolerance, (8) acute gastritis with vomiting, (9) eczema and allied skin conditions, (10) acute ileocoloitis with vomiting, (11) prematurity with defective assimilation, and (12) acute infectious fevers. The stock formula is made as follows: One level tablespoonful of farina to from 5 to 6 ounces 1 per cent. skimmed milk. In very young or premature infants the skimmed milk may be diluted with water half and half. One level tablespoonful of cane sugar or dextrimaltose to 30 ounces of mixture. This cooked together from 30 to 60 minutes, using either a single or double boiler, until the resulting mixture is so thick and tenacious that it will barely fall off an inverted spoon. Thorough cooking is absolutely essential. There is usually some difficulty in getting the baby to take its feedings when first put on thick cereal. This difficulty is overcome by using larger amounts of cane sugar, which makes it more palatable. From 70 to 80 calories for each pound of body weight is divided up among the required number of feedings for the child's age. Each ounce of the mixture contains 24 calories. From 3 to 4 hour intervals may be employed for feeding.

METABOLISM STUDIES ON INFANT GROWTH

Rost [40] studied the effect of the addition of calcium to the food of fifty children. To undertake this study he prepared cakes containing a known quantity of calcium. In order not to increase the food and calories suddenly, two calcium free cakes were given for a seven weeks'

39. Mixsell, H. R.: Three Years' Experience with Cereal Feedings in Infancy in Malnutrition and Allied Conditions, Atlantic M. J. **26**:744 (Aug.) 1923.

40. Rost: The Nutritional Condition of the Children of the Berlin Orphans' Home with Special Consideration of Calcium Metabolism, Arch. f. Kinderh. **72**:81 (Nov. 18) 1922.

preliminary period. Then calcium cakes were given for seven weeks; calcium lactate being used. Two cakes contained a total of 80 gm. These were made up of wheat flour, corn meal, margarine, sugar and spices. Analysis showed sodium 1.04 gm., calcium monoxid 0.035 gm., calcium magnesia 0.074 gm., and fat 7.36 gm. The caloric value was 352. The children were in a good state of nutrition, being only slightly below normal in height and weight as a result of their war experience. After a continuous diet for six weeks, with a daily allowance of 3.6 gm. calcium lactate, corresponding to calcium monoxid 0.77 gm., two tests were made at the beginning and at the end of the calcium period, with considerable retention of calcium.

In taking up this question from a slightly different point of view, Holt [41] investigated the calcium metabolism of seven healthy children between the ages of from 2 to 6 years, first, on the usual hospital diet of whole milk, cereal, bread with butter, meat or eggs, fresh vegetables, potato or rice, and stewed fruit, and later on a reduced fat intake. It was possible to reduce the fat by substituting fat free milk for whole milk, increasing the bread, cereal, fruit, and adding cane sugar. During the fourth day preliminary metabolism period, on a mixed diet, the calcium was normal. The absorption was 0.51 gm. daily or 0.028 for each kilogram. This was very close to the average found by others for healthy children of the same age. Fat absorption was normal and fat excretion in the usual amount and form, as soap, fatty acids and neutral fats. After one week on low fat diet, the total daily amount of solids in the stools had increased from 16.8 to 24.4 gm., after three weeks it was averaging 34.3 gm. daily. Calcium oxid excretion after one week had increased from 1.34 to 1.81 gm. and in three weeks it had increased to 2.22 gm. This was more than the intake or a negative daily calcium oxid balance. In summarizing his observations Holt [41] observed that the stools uniformly change from a smooth, normal alkaline consistency, well digested, to one acid and foul, with an excess of fermentation and undigested food residues. Whether this change was due to the reduction of fat or to increased carbohydrates, or to both factors, it is impossible to say definitely. The increase in calcium oxid excretion was in most cases due partly to the fact that on a low fat diet the daily amount of total solids in the stool was markedly increased. The calcium oxid percentage of total solids in the stool was high. From his observations, the indications are that a low fat diet diminishes calcium oxid absorption. Whether this is dependent on a definite relationship between the fat and calcium in the diet, or whether

41. Holt, L. E., and Fales, H. L.: Calcium Absorption in Children on a Diet Low in Fat, Am. J. Dis. Child. **25**:247 (March) 1923.

the fat intake influences calcium oxid absorption, chiefly by the maintenance of a proper condition, it is impossible to say.

In studying basal metabolism and its influence on growth in young children, Fleming [42] has plotted two curves. These show the yearly increase in weight of a normal child per kilogram, for the first 10 years of life; also the daily basal metabolism per kilogram. These two curves do not correspond very closely, except in the first 2 years of life, they both show the same tendency to flatten out as the age increases and the rate of growth decreases. They apparently afford a certain amount of evidence in favor of high basal metabolism in the growing child, which may be accounted for by the amount of energy expended in the manufacture of new tissue. Quoting the work of Rubner [43] he places the figure of the growing human being at 5.2 per cent. of the calories in the diet, as retained in the form of new tissue; on an average of 27 calories daily during the first half year of life. Calculations have not been published to show how much of the basal heat output is devoted to the manufacture of new tissue.

In investigating the relationship of the course of enteral salt fever and the calcium content of the serum, Schoenfelt [44] believes there is still a great deal of dispute regarding enteral sodium chlorid fever. He believes this is due to a combined action of whey and sugar in the infant's intestine. For the experiments regarding the course of the fever and the chlorin curve, only infants without infections or serious nutritive disturbances were used. From his observations on salt fever he is inclined to make the interpretation that it is a direct elective action of the salt on the heat centers and of the nerve tracts leading to it. In a number of patients the administration of sodium chlorid produced all the symptoms of alimentary dyspepsia, evidently due to injury of the gastro-intestinal function. The irregular and uncharacteristic temperature elevation in these cases distinguishes it from true salt fever. There is a time connection between the fever curve and the blood chlorin curve, from which it is evident that if the salt acts directly to produce fever it must act through the blood. Also in patients in which there is no fever after the giving of salt there is a rapid rise in the blood chlorin curve. The absence of fever can then be explained only on the basis of other factors or complications.

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42. Fleming, G. B.: The Influence of Growth on the Basal Metabolism of Children, Am. J. Dis. Child. **25**:85 (Feb.) 1923.

43. Rubner: Das Problem der Lebensdauer und Seiner Beziehung zu Wachstum und Ernahrung, Ztschr. f. Biol. **36**:1, 1898.

44. Schoenfelt: Theory of Enteral Salt Fever, Experiments on the Relation Between the Course of the Fever and the Calcium Content of the Serum Arch. f. Kinderh. **72**:120 (Nov. 18) 1922.

ANTISCORBUTIC CAPACITY OF DIFFERENT FOOD SUBSTANCES

Lesne [45] has tested guinea-pigs with various milk preparations. Milk boiled for ten minutes produced no scorbutics. A year old, unsweetened, condensed milk sterilized at high temperature produced scorbutus. Aging after sterilization clearly destroys the vitamins. A similar preparation recently sterilized kills the test animal by producing scorbutus. More than 50 per cent. of infantile scurvy is caused by milk that has been rendered homogeneous. Saccharose or lactose is antiscorbutic. Test animals fed with powdered milk die of scurvy in the same time as those fed with sterilized milk. Very few of the prepared milks are capable of protecting infants or guinea-pigs against scurvy. Some infants resist the absence of vitamin remarkably. Orange, lemon or tomato juice should be employed in artificial feeding. Lemaire [46] described the development of scurvy in an infant, aged 9 months, fed on fresh cow's milk. The milk was sent from a farm not far distant; it was subjected to a single heating for ten minutes, before being placed in the nursing bottle, but not modified in any other way. Cure was readily obtained with lemon juice. He called attention to the fact that this disease is relatively not infrequent, even though milk is heated twice or more. Leichtentritt,[47] in a clinical experiment on this subject, has tried to determine the effect of certain substances on lemon juice. Lemon juice boiled for one hour does not interfere with growth. Exposing lemon juice to ultraviolet rays or passing oxygen through it does not change its effect. When it is allowed to stand for several days the antiscorbutic action is lessened.

OTHER TYPES OF FOOD USED IN INFANT FEEDING

McIlwaine [48] advocates the use of dried milk as one of the weapons with which to combat digestive disturbances in infants, or to tide over a critical period. Properly prepared it is sterile and will keep indefinitely. It is not easily contaminated even by the most ignorant. In traveling it is a great help, as its preparation is so simple. He takes exception to the French idea that if milk powders are kept too long scurvy may result. He has found, on the contrary, that they may be used after several months or even a year's time. Chemically, they are not comparable to fresh milk, although they are very superior

45. Lesne, E.: Antiscorbutic Capacity of Different Milks Employed for Infant Feeding, Nourrisson, Par. **10**:377 (Nov.) 1922.

46. Lemaire, H.: Barlow's Disease in an Infant Fed on Fresh Cow's Milk, Bull. Soc. de pédiat. de Par. **7**:329 (Oct. 17) 1922.

47. Leichtentritt, B.: Clinical and Experimental Studies on Barlow's Disease, Ztschr. f. d. ges. exper. Med. **29**:658 (Oct. 18) 1922.

48. McIlwaine, M. N.: Virginia M. Monthly **49**:700 (March) 1923.

to a good many specimens of fresh cow's milk. In an analysis of fifty cases he shows some very good results. Dryco was the milk powder employed.

Muller [49] has described the use of potato flour in feeding young children. This has been brought about by the restriction of fresh food in Berlin; the population was required to use potato flour and other forms of starch. Ordinary potato flour, being pure starch, should not be employed. This can be overcome by roasting the flour, which partly converts the starch. The vitamin content of potatoes is not large, so that it suffers still further in the drying of the potatoes and the roasting of the flour. It is higher, however, than the vitamin content of other flours recommended for child feeding. There are no digestive disturbances on changing to this flour. The slightly laxative action is often an advantage. The stools are somewhat more solid than when wheat flour is employed. The urine shows a leaning toward the alkaline side, while in most infants flours convert the reaction to an acid one. According to current theory potato flour is indicated in rickets and contraindicated in tetany.

49. Muller, E.: Potato Flour in the Feeding of the Small Child, Klin. Wchnschr. **1**:2378 (Nov. 25) 1922.

News and Comment

The French Association of Pediatrists will hold its fourth Congress for French speaking Pediatricians, September 29, 30 and October 1, 1924, at Paris, under the presidency of Dr. Marfan. The following questions are to be reported on:

First: Etiology and Pathogenesis of Rickets, by Lesné.

Second: Prophylaxis and Treatment of Syphilis in Childhood, by L. Tixier.

Third: The Treatment of Empyema in Childhood, by Ribadeau-Dumas and Rocher.

Abstracts from Current Literature

Anatomy and Physiology

FURTHER OBSERVATIONS UPON THE SHADOWS OF THE THYMUS AND THE HEART. L. R. DEBUYS and E. C. SAMUELS, South. M. J. **17**:260, 1924.

Four hundred and fifty observations were made on newly born infants and infants aged 6 months and 1 year. All the observations were true antero-posterior exposures. The study shows that the heart apparently does not rotate during the first year as the individual grows, but the shape remains the same as to type and position. There is an increase in its size. The great blood vessels of the mediastinum show a decided lengthening during the first year period.

The growth of the size of the heart is not influenced by the size or position of the thymus.

The shadow of the thymus does not gradually increase or decrease uniformly in the healthy infant within the first year of life. It may vary in size in the same person at different periods within the first year. The variations in the size of the shadows may exist without clinical evidence of disease.

SCHLUTZ, Minneapolis.

RELATION OF FLEXORS TO EXTENSORS IN NEW-BORN. C. RITTER, Jahrb. f. Kinderh. **104**:293, 1924.

So far as weight is concerned, the relation of flexors and extensors of the extremities is practically the same in infants and adults. The flexion of the extremities in new-born infants, therefore, is not due to greater development of flexors. It is more likely that reflex nervous relationships are the cause.

SUTHERLAND, Chicago.

Pathology and Bacteriology

THE INTRADERMAL SALT SOLUTION TEST. II. ITS PROGNOSTIC VALUE IN "NEPHRITIS" WITH GENERALIZED EDEMA. C. A. ALDRICH and WILLIAM B. MCCLURE, J. A. M. A. **82**:1425 (May 3) 1924.

It was found that the elevation produced by the injection of 0.2 c.c. of 0.8 per cent. sodium chlorid solution disappeared in normal children of 1 year of age, or over, in sixty minutes or longer.

Since the disappearance time sharply decreases coincident with or preceding the development of edema, and increases with or before the subsidence of edema, the curve of the disappearance time has been used as an index of the tendency to increasing or decreasing edema and as a means of ascertaining a return to normal.

The report of five cases substantiates the interpretation of the disappearance time curves. The observations made seem to confirm the theory that the tissues take an active part in the production of edema.

HENN, Chicago.

HISTOLOGICAL STUDIES ON THE IRON METABOLISM IN EARLY INFANCY. P. SCHWARTZ, R. BAER and J. WEISER, Ztschr. f. Kinderh. **37**:167, 1924.

One hundred and fifteen infants were examined at necropsy and histologic studies were made of the livers and spleens. One group of thirty-eight consisted of stillborn infants and infants under 3 days of age. Ten per cent. of

these infants were entirely free from iron in both liver and spleen as demonstrated by tissue stains. The remainder showed traces of iron in both organs. In the older infants increasing amounts of iron were found. This increase was found first in the periportal, endothelial tissues and in the red pulp of the spleen; later the iron was more generally distributed. The iron content of the liver and spleen ran parallel. The Küpffer cells appear several days after birth. At from four to ten weeks the liver and spleen show the maximal amounts of iron. After five months there is a gradual decrease of iron and at eight months or earlier both organs are again iron free.

ORGANIC ACIDOSIS IN INORGANIC ACIDOSIS, AND ALKALOSIS. H. BEUMER and ANNA SOECKNICK, Ztschr. f. Kinderh. **37**:236, 1924.

In acidosis following starvation, sodium bicarbonate and secondary sodium phosphate hastened the acetonuria; ammonium chlorid delayed it. The explanation of the action of these salts may be found in their effect on glycogen mobilization in the liver. Alkalosis favors the appearance of acetonuria in tetany, pneumonia, and lye poisoning.

THE ENDOTHELIAL SYMPTOM IN INFANTS. CLARA BEINTKER, Ztschr. f. Kinderh. **37**:225, 1924.

The Rumpel-Leede phenomenon was studied in 400 infants under 1 year of age and 100 children between 2 and 3 years of age. The tourniquet was kept on the arm from four to five minutes. The reaction (intracutaneous capillary hemorrhage) occurred in the first three minutes. In the 400 infants the phenomenon was positive in 36 per cent., in the 100 older children it was positive in 44 per cent. In the first three months the reaction was seldom seen. The phenomenon was observed in seven of ten cases of measles, in eight of twenty-three cases of pertussis, in 65.2 per cent. of twenty-three infants with syphilis, in 61.5 per cent. of thirteen tuberculous children, in 70 per cent. of twenty-four children with spasmophilia, and in 82 per cent. of fifty-six children with exudative diathesis. Two factors play a rôle in this endothelial symptom: (1) Constitutional diathesis; (2) nutrition.

PETERMAN, Rochester, Minn.

HAEMOLYTIC AND AGGLUTINATIVE PROPERTIES OF DIPHTHERIA ANTITOXIN (Preliminary Report). HOWARD OSGOOD, Bull. Buffalo Gen. Hosp. **2**:22, 1924.

Osgood believes that the reactions following the use of New York State Antitoxin intravenously, were not due to "tricresol laking." The presence of blood serum in great excess, he thinks, caused an inhibition of the laking. In his laboratory experiments, he also found slight agglutination of human erythrocytes by diphtheria antitoxin in 1:4 dilution.

This weak agglutinative activity, he thinks, would have no effect on the patient's erythrocytes when the antitoxin is injected intravenously.

FOUR CASES OF CONGENITAL DIAPHGRAGMATIC HERNIA IN THE NEW-BORN. W. F. JACOBS, Bull. Buffalo Gen. Hosp. **2**:26, 1924.

Jacobs adds four more cases of congenital diaphragmatic hernia to the series of 600 cases recently reported by Eppinger. He reports one case of aplasia of the esophagus, which is extremely rare.

A CASE OF OPEN MECKEL'S DIVERTICULUM WITH PROTRUSION OF EVERTED BOWEL THROUGH THE UMBILICAL CORD IN THE FORM OF A HERNIA SAC FILLED WITH LOOPS OF INTESTINE. THEW WRIGHT, Bull. Buffalo Gen. Hosp. **2**:30, 1924.

Wright reports an uncommon congenital condition due to faulty closure of the vitello-intestinal duct.

BACON, Chicago.

THE USE OF RABBITS FOR INTRACUTANEOUS VIRULENCE TESTS OF B. DIPHTHERIAE OR TITRATION OF DIPHTHERIA ANTITOXIN. D. T. FRASER and H. E. WIGHAM, J. A. M. A. **82**:1114 (April 5) 1924.

Rabbits of 2 kg., or more, in weight, with unpigmented skin are used; the back and flanks are clipped closely; this area is covered lightly with a thin paste of barium sulphid and starch 1:4; quickly and thoroughly washed off with soap and water, immersed in warm 0.85 per cent. saline solution and dried. The animals are prepared twelve hours before the test. Areas of approximately 3 by 3 cm. are outlined with a moistened indelible pencil. The injections of 0.1 c.c. are made with a 26 gage, short bevel needle, and syringe graduated in fiftieths of a cubic centimeter.

The skin reactions are comparable with those obtained in the skin of guinea-pigs. In rabbits, however, the areas are greater in extent and develop more slowly; necrosis, when present, is more superficial. The maximum reaction appears as a rule on the third day or even later. The minimal lethal dose of the toxin used in the authors experiments was found to be 0.005 c.c. for 250 gm. guinea-pigs; the L + dose of 0.38 c.c. contained seventy-six minimal lethal doses. One fifteen hundredth of the minimal lethal dose contained in 0.1 c.c. produces a very definite redness with scaling on the fourth day.

HENN, Chicago.

PREPARATION OF DRY ANTITOXIN AND AGGLUTININ POWDERS. EDWIN F. HIRSCH, J. Infect. Dis. **34**:390 (April) 1924.

Methods involving the hydrogen-ion control for the preparation of dry antitoxin and agglutinin powders are described. Dry powders of the antitoxins, in all probability, may be prepared from specific immune serums according to the method outlined.

The dry powder, with active agglutinating properties was prepared from rabbit and human plasma. It is a protein-acid salt, readily soluble in water, and in solution actively potent.

Agglutination has been obtained with mixtures of homologous bacteria in dilutions containing as little as 0.0035 mg. and 0.0022 mg. of dry weight per cubic centimeter.

One of the preparations has been stored about five weeks without loss of potency.

KENNEDY, Rochester, Minn.

EXPERIMENTS ON THE SURVIVAL OF THE FEBRILE HERPETIC AND ALLIED VIRUSES IN VITRO. JAMES E. MCCARTNEY, J. Exper. Med. **39**:533, 1924.

The author studied the survival and multiplication of the so-called virus of epidemic encephalitis and the virus of febrile herpes in artificial cultures. His experiments fail to confirm the statements previously made by Bradford, Bashford, and Wilson, by Loewe and Strauss, and by Thalhimer, that microorganisms of the class of the globoid bodies of poliomyelitis may be cultivated by the use of the Smith-Noguchi anaerobic tissue method from the so-called virus of encephalitis lethargica. The experiments show equally that the herpes virus does not multiply in this medium. They indicate, moreover, that the medium is unfavorable to the survival of the virus, while ordinary broth under aerobic conditions is more favorable for maintaining the activity of both of the viruses.

It has also been shown that the viruses can be passed from brain to brain in the rabbit through a long series, without contamination with cocci or other common bacterial forms. Hence the author regards all reports of the finding of ordinary bacteria in the brain of cases of epidemic or lethargic encephalitis as instances of mixed or secondary infection arising during life, or examples of necropsy invasion of the body, or a faulty technic at necropsy.

EXPERIMENTAL HUMAN INOCULATIONS WITH FILTERED NASAL SECRETIONS FROM ACUTE CORYZA. ROBERT C. ROBERTSON and ROBERT L. GROVES, J. Infect. Dis. **34**:400 (April) 1924.

The nasal secretions of eleven patients suffering with acute coryza were collected, diluted with normal salt solution and filtered through a Berkfeld filter. Cultures were made from each filtrate. All cultures were negative after ninety-six hours aerobic and anerobic incubation. On smears made from the filtrates, coccoid bodies were found.

Spraying the filtrates on the nasal mucosa of one hundred healthy persons was followed, for a period of twelve days, by upper respiratory or bronchial affections in only five cases. These cases were considered due to causes other than the inoculations.

During an acute attack of coryza, definite variations were noted in the bacterial flora of the secretions. During the onset and early stages of the attack, there was a marked diminution of the total bacterial flora, with an equally marked predominance of one of the normal habitants—usually staphylococcus albus.

During the purulent stage of the attack, a marked increase of all organisms over the normal flora of health was observed although the predominance of one organism still remained. The later stages of the attack were marked by a gradual return to the normal flora of health.

KENNEDY, Rochester, Minn.

THE EFFECT OF DIGESTIVE JUICES ON THE POTENCY OF BOTULINUS TOXIN. JACQUES J. BRONFENBRENNER and J. MONROE SCHLESINGER, J. Exper. Med. **39**:509, 1924.

Botulinus toxin is a notable exception to the general rule that true toxins from cultures of pathogenic micro-organisms tend to be harmless when taken by mouth. The marked toxicity of botulinus toxin when ingested brings up the question of its path of absorption and its resistance to gastric and intestinal secretions. Landman found that it resisted exposure to fifth normal hydrochloric acid solution for twenty-four hours, but neither he nor other earlier investigators took into account the buffer content of the vehicle fluid in which the toxin is present.

The authors investigated the effect of changes of hydrogen-ion concentration (to which the toxin is subject in passing through the stomach and intestines) either alone or combined with the effect of the digestive ferments. The potency of the toxin was tested by intraperitoneal injection into mice. They conclude that botulinus toxin resists a degree of acidity equivalent to that of the stomach, even when exposed thereto for twenty-four hours at 37 C. In a medium of weakly alkaline reaction, however, its potency is reduced to less than one-tenth in twenty-four hours. It is unaffected by peptic and tryptic digestion. Because of its instability in mildly alkaline solutions and resistance to acids, there is reason to believe that the toxin is in the main absorbed from the upper portion of the digestive tract, i. e., the stomach and upper duodenum.

MCCLURE, Evanston, Ill.

THE EFFECT OF ANESTHESIA AND OF SEDATIVES ON THE SERUM THERAPY OF EXPERIMENTAL BOTULISM. JAQUES J. BRONFENBRENNER and HARRY WEISS, J. Exper. Med. **39**:517, 1924.

The rapid progress of the intoxication in severe cases of botulinus poison in man has rendered of questionable value the therapeutic use of antitoxin in such instances. The time required for making the diagnosis and the delay in obtaining the antitoxin consequent on the few points of distribution, usually results

in the use of the antitoxin too late. More numerous and widespread sources of supply of antitoxin are desirable. At the same time a procedure was sought to retard the progress of the intoxication sufficiently to allow more time for the effective administration of antitoxin. The effect of various anesthetics and sedatives was tried out in this respect on guinea-pigs. The authors found that when guinea-pigs are fed large amounts of botulinus toxin they develop symptoms of intoxication within six hours and die usually within twelve hours. Introduced intraperitoneally in large amounts the animal may show symptoms of intoxication at the end of the first hour and die usually within two hours.

If these animals are placed under anesthesia following theadministration of toxin, the intoxication proceeds much more slowly. Anesthesia, by ether, is effective whether the toxin is given by mouth or intraperitoneally. Life in the guinea-pigs kept under ether after the administration of lethal amounts of toxin is prolonged by a period approximately equal to that during which ether anesthesia is continued. When anesthesia is discontinued, the intoxication proceeds at its usual rate. Anesthesia delays the progress of intoxication not only when administered immediately after the intake of toxin, but when administered much later after the intoxication had progressed far enough to cause definite objective symptoms of poisoning. At this late stage of intoxication, the unsupported serum therapy of botulism in guinea-pigs usually remains without effect. If the animals are anesthetized at this time and kept under the influence of ether for some time, antitoxin therapy becomes effective. Indeed the antitoxin treatment can be delayed further for several hours, provided the animals are kept under anesthesia during the interval. Results of similar nature were obtained with luminal sodium, nitrous oxide-oxygen mixture, and morphin used in place of ether.

McCLURE, Evanston, Ill.

EXPERIMENTAL POLIOMYELITIS IN RABBITS. MARY NEVIN and FLORENCE R. BITTMAN, J. Infect. Dis. **34**:420 (April) 1924.

A 5 per cent. unfiltered suspension of poliomyelitis virus, obtained from the Rockefeller Institute, was inoculated intracerebrally, intraperitoneally and intravenously, or by a combination of these methods, into young rabbits. Of the series of rabbits used, only one developed symptoms suggestive of the disease. The clinical condition of the rabbits and the examination of the central nervous system indicated that the effort to produce the disease by this means was unsuccessful.

SPONTANEOUS RECOVERY OF RABBIT FROM EXPERIMENTAL RABIES. RIGNE D'AUNOY, J. Infect. Dis. **34**:425 (April) 1924.

A case of spontaneous recovery of a rabbit from rabies, after full development of symptoms following intracerebral injection of fixed dessicated virus, is reported. Saliva from this animal obtained sixteen days after inoculation, when injected intracerebrally into rabbits produced typical rabic symptoms with Negri bodies in brain sections. Forty days after inoculation the animal's saliva was no longer infectious. Blood serum secured seventeen and ninety days after injection showed no appreciable antirabicidal action.

SPONTANEOUS ENCEPHALITIS IN RABBITS. ERNEST W. GOODPASTURE, J. Infect. Dis. **34**:428 (April) 1924.

Large gram-positive bacillary organisms have been demonstrated in the cerebral lesions of spontaneous encephalitis of rabbits. The cerebral lesions are vascular in distribution and are the manifestation in the brain of a generalized infection, which may produce similar lesions in the lungs and kidneys in which morphologically identical organisms have been found.

STREPTOCOCCI IN RELATION TO ETIOLOGY OF EPIDEMIC ENCEPHALITIS. EDWARD C. ROSENOW, J. Infect. Dis. **34**:329 (April) 1924.

Rosenow reports the results obtained in an extensive study of the relation of streptococci to experimental encephalitis. Consistent results have been obtained in a series of undoubted cases of encephalitis, representing a wide range of types of the disease, and extending over a period of four years. Somewhat peculiar streptococci, much alike in the different cases, have been isolated constantly from infected tonsils, teeth or nasopharynx during life, and from the brain after death. With this streptococcus in freshly isolated cultures, after as high as forty-four rapidly made subcultures, and after a series of animal passages, characteristic symptoms and lesions of different forms of encephalitis have been reproduced in animals.

Peculiar neurotropic properties of the streptococcus have been noted in four species of animals, rabbit, monkey, mouse and guinea-pig, following various methods of inoculation. Intracerebral inoculation, the method that has been so successfully used in virus studies in poliomyelitis, has given the best results. Results such as in these experiments were not obtained with green-producing streptococci from sources other than encephalitis. The organism has been demonstrated in the lesions which develop spontaneously in man, and which have been produced experimentally in animals, and proved absent in adjacent normal tissues and in the brains of persons and animals that died from other causes. It has been shown to possess specific antigenic properties. Most of the strains are immunologically alike, as determined by agglutination experiments with hyperimmune serums. The serum of patients with acute forms of the diseases agglutinated specifically the homologous and many heterogeneous strains. The requirements for proof of the causal relationship between this streptococcus and encephalitis seem to have been fulfilled.

The organism varied greatly in size and shape, depending apparently on conditions of growth. Filtrates of nasopharyngeal washings, of emulsions of the brain in fatal cases, and of old anerobic cultures have often yielded the streptococcus in culture and in positive animals. Large and extremely small forms sometimes in the same chain, and large oval forms breaking into small forms have frequently been seen in cultures and have been demonstrated repeatedly in the lesions in undoubted cases of encephalitis.

KENNEDY, Rochester, Minn.

METHODS AND SIGNIFICANCE OF TESTS OF STOMACH FUNCTION. FRITZ MÜLLER, Monatschr. f. Kinderh. **27**:415, 1924.

All previous methods of stomach examination such as roentgen ray and even chemical examination of stomach contents are inexact, because the emptying time of the stomach and the acidity of the stomach contents are in close relationship.

Formerly, the acidity of the stomach contents was determined by titration, now the hydrogen-ion concentration is determined. A variety of test meals have also been suggested, but many of these do not consider the buffer action of the salts.

Müller illustrates this by the following experiment. He gave a mixture of milk and sodium hydroxid having a p_H of 10. After thirty minutes, 40 c.c. of secretion was removed with a p_H of 7.7. After one hundred and twenty minutes, 48 c.c. with considerable mucus, was removed with a p_H of 6.2.

In another experiment, he gave a mixture of milk and hydrochloric acid, the mixture having a p_H of 4.15. After thirty minutes, 3 c.c. of secretion was removed with a p_H of 4, after one hundred and twenty minutes, 13 c.c. of gastric juice was removed, having a p_H of 3.3.

On account of these observations Müller concludes that neither tea nor gruel should be used for test meals, for: (1) they exert unphysiologic irrita-

tion; (2) their emptying time is irregular; (3) the rapidly increasing acidity of the secretion may keep down the amount.

The most satisfactory test meal he has found is a mixture of equal parts skimmed milk and oatmeal gruel. Of this mixture, to a baby weighing 3,500 gm, he gives 120 c.c. For a baby weighing 5,000 gm., he gives 160 c.c., and for babies weighing over this he gives 200 c.c.

The stomach contents should be removed in one hour.

BASAL METABOLISM IN CHILDHOOD. FRITZ B. TALBOT, Monatschr. f. Kinderh. **27**:465, 1924.

Benedict and his co-workers have been able to construct a respiratory chamber which permits the estimation of both carbon dioxid and oxygen. As this apparatus is practicable for experiments as short as one-half hour, much advance in the study of metabolism has been made. Technically speaking, basal metabolism requires the subject to be at absolute rest with the stomach empty. As this is impossible in infants, these subjects have been investigated during the quiet period following a feeding. Howland has proved that the indirect method is just about as accurate as the direct measuring of heat given off by the body.

That muscular activity increases basal metabolism has been investigated by Rubner and Heubner in 1898, and confirmed by Howland in 1912. Benedict and Talbot, in carefully controlled experiments, showed that metabolism was increased by muscular activity from 30 to 40 per cent., and in one very restless crying child by 211 per cent.

Benedict and Talbot, as well as Bailey and Murlin, confirm the original observations of Hesselbach that basal metabolism in the new-born is very low and increases proportionally to age. In a like manner, the respiratory quotient falls rapidly from birth to 0.73 on the third day, showing that the glycogen supply has been exhausted and that a large part of the body energy is supplied by the fat. In premature and the new-born infants a relation exists between heat production and body weight, the smaller the child the lower the heat production. However, the metabolism of the premature infant is probably greater than that of the fetus of the same weight, due to the demands of extra-uterine life. The metabolism of the premature infant is very slight in comparison to the normal infant.

In normal and older infants growth exerts a marked influence on metabolism. The curve of growth and heat production run nearly parallel for the first year, but after that, results are not definite.

Many observers agree that total heat production increases with age. The same relation applies to increase of weight, confirming Benedict and Talbot's theory that heat production depends on the active protoplasmic tissue of the body. This relationship is not so constant in girls, perhaps due to varying amounts of fat.

As regards the metabolism per kilogram of body weight with reference to age, this increases for the first year, and then gradually decreases. Fat children have lower metabolism than the thin ones.

Of children over 11 kg. in weight, girls have lower metabolism than boys, possibly due to their having less "active tissue" and more fat.

At puberty there is an increase, perhaps due to increased thyroid activity.

Food increases metabolism from 8 to 15 per cent. The latter is difficult to estimate in infants.

In disturbances of nutrition, the farther under the normal weight, the greater the child's metabolism, the baby probably using a larger proportion of body tissue for warmth.

The study of metabolism is of clinical value not only in feeding cases but in endocrine disturbances. It is markedly decreased in cretinism and can be used as a guide to the dosage of thyroid extract. The same applies to other

types of thyroid disturbance. In mongolian idiocy no change was found. Studies have also been made on individual cases of dwarfs, congenital brain defects, dyspituitarism and amaurotic family idiocy.

Nutritional Disturbances

CONCERNING THE TECHNIC OF THE USE OF PROTEIN MILK. PAUL, Monatschr. f. Kinderh. **27**:504, 1924.

It is now generally admitted that dyspeptic stools occur not only in cases of intestinal fermentation with acid bowel movements, but also with alkaline bowel movements, suggesting that the two processes, fermentation and putrefaction, go on simultaneously. As putrefaction in the intestine is associated with an increase of indican in the urine, Paul believes that the determination of the amount of indican in the urine would be an index of the predominance of fermentation or putrefaction in the intestine, and thus a guide as to the increase or decrease of carbohydrate.

Paul followed this procedure, and in those patients with stationary weight and indicanuria, in spite of dyspeptic stools, he increased the carbohydrate. These patients showed prompt response in the weight curve, the general condition improved and the bowel movements became normal.

Paul believes that this little test will prove a useful clinical guide to the dosage of carbohydrate.

ACTION AND EFFECT OF HYDROCHLORIC ACID MILK IN TETANY. R. SCHEER and A. SALOMON, Monatschr. f. Kinderh. **27**:406, 1924.

Scheer has previously reported the beneficial effect of hydrochloric acid on tetany, that is, he has shown that the influence of this acid causes disappearance of mechanical and electrical hyperexcitability. Many observers have disputed these conclusions with a suggestion that the syndrome may be due to a number of etiologic factors.

The authors determined to repeat their observations not only in clinical experiment, but also by determining the serum content of calcium and phosphate. Previously, Scheer had concluded that hydrochloric acid caused an increased phosphate excretion by the kidney.

The first experiments were made with a mixture of milk, 740 c.c., one-tenth normal hydrochloric acid, 260 c.c., 5 per cent. sugar. While many cases seemed to improve greatly under this mixture, a few of the very severe cases became worse. For the latter, they tried increasing the strength of the mixture to milk 600 c.c., tenth normal hydrochloric acid 400 c.c., giving a *p*H of from 3.5 to 4. Even the severely ill patients showed striking improvement on this mixture.

As regards the serum analyses, the phosphorus was decreased and the calcium increased.

This milk mixture seemed also of some value in cases where the bowel movements were very loose.

GERSTLEY, Chicago.

SCURVY IN CHILDHOOD. B. SCHAGAN, Jahrb. f. Kinderh. **104**:225, 1924.

In the years following the famine of 1918-1919, there was a great increase in cases of scurvy throughout Russia. Whereas in 1913 there were only 7.3 cases per 10,000 inhabitants in St. Petersburg, in 1920-1923, from 5.2 to 11.3 per cent. of the cases in the children's clinic were scurvy patients. The observations cover a series of 186 cases, 55 per cent. of which occurred in the spring and 32 per cent. in the summer. The highest incidence was in children from 2 to 8 years of age. The diet had a sufficient caloric content but was almost lacking in the antiscorbutic factor C.

Scurvy is a severe toxicosis with involvement of many organs, and a high mortality, 16 per cent., in the period studied. The stomatitis became gangrenous in 13 per cent. of the cases. The morphology of the blood and roentgen rays show a severe disease of the bone marrow. In this series 72 per cent. of the cases had a strongly positive Pirquet reaction, and 15 per cent. had a weakly positive reaction. This is taken to indicate that a latent tuberculosis is activated by scurvy. The general treatment, consisting of fresh vegetables and raw milk, gave good results, and the stomatitis was best treated locally with a 1 per cent. copper sulphate solution.

SUTHERLAND, Chicago.

NOTE OF THE CLINICAL SIGNS OF INFANTILE RICKETS AS OBSERVED IN VIENNA. H. P. WRIGHT, Canad. M. A. J. **14**:320 (April) 1924.

This article is a review of Part III, No. 3, special report series No. 27, of the Medical Research Council of Great Britain, 1923, by E. J. Dalywell and Helen M. M. Mackay.

The author concludes that craniotabes is practically always a sign of active rickets, and that it is an early sign.

The rachitic rosary is an important and early sign, but epiphyseal enlargement and delayed closing of fontanels are not considered of much importance in the early diagnosis. All other signs are considered variable. They divide rickets into two types: (1) rickets with low blood calcium and normal phosphorus and (2) rickets with low blood phosphorus and normal calcium.

MOORE, Omaha.

Diseases of the New-Born

CONGENITAL GOITER. H. H. SKINNER, J. A. M. A. **82**:1190 (April 12) 1924.

In twelve cases of congenital goiter, reported by Skinner, he believes that they are caused by iodin deficiency in the mother's diet. All grades of pathologic change may be seen, from a simple parenchymatous goiter with no symptoms, to marked enlargement with complete tracheal compression and cystic conditions causing serious danger to mother and child. Treatment is surgical for emergencies but mainly prophylactic by means of iodin administration to the mother.

LIGATION OF THE UMBILICAL CORD BEFORE THE CESSATION OF PULSATION. A. C. WILLIAMSON and J. V. TAYLOR, J. A. M. A. **82**:1107 (April 5) 1924.

The authors found that there was no appreciable difference in the weight or progress during the first 2 weeks in life, between babies whose umbilical cords were cut after cessation of pulsation and babies delivered by the cesarean method, whose cords were ligated immediately following delivery. Late ligation should not be urged on the ground that the infant thereby gains an extra appreciable amount of blood.

HENN, Chicago.

MENINGEAL HEMORRHAGES OF THE NEW-BORN. ALFRED GORDON, Am. J. Obst. & Gynecol. **7**:462, 1924.

The causes of meningeal hemorrhages are largely the result of tearing of the blood vessels in the cerebral membranes during labor. This is most marked in forceps and breech deliveries, especially if the delivery is terminated too rapidly or if the forceps are applied to the long diameters of the head. Due to the anatomy of the fixed point in the vein of Galen when the apex of the tentorium is drawn up, the tributary veins rupture. For this reason nearly half of the meningeal hemorrhages occur in the tentorium.

The author divides the hemorrhages into supratentorial and infratentorial. In the former, the blood cannot extend beyond the lower surface of the tentorium; in the latter, it may extend to the spinal canal. He advises spinal puncture in each case as a diagnostic procedure for localization of the lesion and treatment of the infratentorial. Early craniotomy in the supratentorial is directly indicated, and favorable results can be expected when the operation is performed within a few days after birth.

HEMATOCOLPOS IN A CHILD OF SIX WEEKS. G. L. MOENCH, Am. J. Obst. & Gynec. **7**:471, 1924.

The author reports a case of hematocolpos in a child, aged 6 weeks. The child was found dead in bed. There was no history of cough, fever, or illness.

Necropsy findings were normal except for the presence of a large patent foramen ovale, and some old tarry blood on the anterior parietal peritoneum which had evidently come from a small cyst of the right ovary. The hymen was imperforate and the vagina was dilated to the size of a golf ball by a collection of old blood. There was marked pulmonary edema, although the lungs were not fully expanded. The cervix was slightly eroded, but there was no evidence of a hemorrhagic diathesis. Probable diagnosis was either precocious menstruation or acute inflammation with hemorrhage.

ALLEN, Chicago.

Acute Infections

A SCARLET FEVER ANTITOXIN. GEORGE F. DICK and GLADYS HENRY DICK, J. A. M. A. **82**:1246 (April 19) 1924.

With the idea of producing a scarlet fever antitoxin, a horse was immunized by subcutaneous injections of sterile filtrate, from broth cultures of strains of streptococci that had produced experimental scarlet fever. Part of the horse serum was concentrated and tested in various dilutions. The tests revealed that the antitoxin had not been destroyed.

It will be necessary to determine the therapeutic value by the use of the antitoxin in a large series of carefully controlled cases.

THE RELATION OF THE DICK TEST TO SCARLET FEVER. C. F. BRANCH and F. G. EDWARDS, J. A. M. A. **82**:1260 (April 19) 1924.

Branch and Edwards, using streptococcus filtrate, made by the Dicks, found that: all convalescent scarlet fever patients tested gave negative reactions, that some patients ill with scarlet fever gave slightly positive reactions which became negative in the convalescent period two weeks later. The filtrate with which the Dicks produced 41.6 per cent. positive or strongly positive skin tests gave 40 per cent. positive tests in the hands of the authors. The control used was negative in all cases tested.

THE PROPHYLAXIS OF VARICELLA WITH CONVALESCENTS' SERUM. A. A. WEECH, J. A. M. A. **82**:1245 (April 19) 1924.

Nine infants exposed to varicella were given from 3.0 to 4.5 c.c. of convalescents' serum, intramuscularly, within from one to six days after exposure. Eight of them did not contract the disease. In the ninth infant an extremely mild attack developed after an unusually long incubation period of twenty-two days. The serum used was obtained from children in whom the disease had developed from ten to twenty days previously. Weech suggests the use of this serum in hospital practice, thus diminishing the period of quarantine.

FURTHER NOTES ON THE TREATMENT OF PERTUSSIS BY THE ROENTGEN RAY. H. I. BOWDITCH, J. A. M. A. **82**:1422 (May 3) 1924.

In three hundred cases of whooping cough treated by the roentgen ray, there was strong evidence that more than 80 per cent. were benefited by the treatment. Only one fatal case occurred. The most favorable results were obtained in the early stages of the disease and in the younger children.

The justification of the roentgen-ray therapy is based on the present theory of probable involvement of the hilum lymph nodes in an acute inflammatory hyperplasia, and the action on these glands in reducing their size.

The ordinary dosage for a child aged from 5 to 10 years was five minutes exposure with a Coolidge tube set at 4.0 ma., a 1 mm. aluminum filter and a 6 to 7 inch back up spark at a distance of 28 inches.

Treatments were given every alternate day for three treatments, followed by a second course beginning seven days later. Relief usually followed a few hours after the third treatment. Very few patients had more than four treatments.

HENN, Chicago.

EPIDEMIOLOGY OF DIPHTHERIA. HANSSEN, Jahrb. f. Kinderh. **104**:201, 1924.

Diphtheria appeared in Schleswig-Holstein in the first part of the nineteenth century, though it did not become an epidemic until 1860. It appeared first in the southern part and spread northward. It was distinctly a children's disease, and has remained so, the most susceptible age being from 1 to 5 years. This rather extensive study bears out the usual ideas, namely, that diphtheria is a disease of populated centers and is spread by contacts in school, church and other crowded buildings. The serum treatment has greatly reduced the number of cases and the mortality since 1894.

STUDIES ON THRUSH. BERTHOLD EPSTEIN, Jahrb. f. Kinderh. **104**:129, 1924.

This is an extensive study of thrush, particularly to determine the latent form of the thrush fungus, and its relation to the acute disease. In addition to microscopic and cultural characteristics he was able to immunize rabbits and use a complement fixation test to aid in differentiation of types. The serum of children with thrush did not, however, give the complement fixation reactions.

Of 2,000 infants, active thrush was found in 270 cases, but none during the first 5 days of life. The most occurred during the second week, and few in the second half year. Eighty-one per cent. of the cases were distinctly ill. Latent thrush was much more frequent during infancy than the active disease, and was found as early as the second day of life.

A larger percentage of pregnant women and women with children under 1 year of age have thrush, than have those not pregnant or with only older children. In all cases of thrush in the infant, fungi of the same variety were previously present in the mouth of the mother. If the mother's mouth was free of fungi, the child remained free. If the child's mouth was infected, the fungus appeared next on the breast of the mother and later in the child's stool.

Latent organisms in the mouth may cause active disease if the infant is very young, premature, weak or very icteric, or the thrush may remain latent for months and become active with the onset of an acute disease not related to thrush. Attempts to change a latent to an active thrush in new-born kittens by local application of hydrochloric acid solution and trichloracetic acid were not very successful, though they did succeed in one case. The prophylactic treatment suggested is that the mother should use a mouth wash before and soon after delivery.

SUTHERLAND, Chicago.

PYREXIAS OF OBSCURE ORIGIN IN CHILDREN. PHILIP F. BARBOUR, South. M. J. **17**:256, 1924.

Intestinal toxemias are common causes of pyrexias. In the earlier stages, these conditions are not easily differentiated from typhoid and paratyphoid. Pneumonia can be masked by pronounced intestinal symptoms. Pyelitis is frequently overlooked. It is one of the commonest causes of pyrexias. Repeated urine examinations are absolutely essential to the diagnosis. Focal infections play a large part in the production of obscure pyrexias. The tonsils are apparently the chief seat, but sinus trouble must not be overlooked and is more often responsible for pyrexias than is commonly supposed. Tuberculosis must always be thought of and its presence or absence definitely established.

SCHLUTZ, Minneapolis.

CONVALESCENT WHOLE BLOOD, PLASMA AND SERUM IN PROPHYLAXIS OF MEASLES. ABRAHAM ZINGHER, J. A. M. A. **82**:1180 (April 12) 1924.

Of one hundred and two children receiving convalescent whole blood, plasma or serum, ten developed measles. Of these, seven were injected on the eighth day of exposure and developed a modified form of measles after twenty-two days. One child injected on the seventh day of exposure developed typical measles twenty days later, after a period of incubation of twenty-seven days. The tenth child was protected by 5.0 c.c. of convalescent serum for twenty-eight days, when she was exposed to a second case of measles and developed a typical attack fourteen days later, forty-two days after receiving the serum.

Of the five control children, four developed typical measles after periods of incubation varying between twelve and fifteen days.

Of fifty-eight additional children, injected in two institutions, twenty-three developed a mild and modified form of measles and three a typical form of the disease with characteristic catarrhal symptoms. In these three children, a thirty-five day convalescent plasma was used in doses of 3.0 c.c. Of thirty-three control children, three showed a mild form and ten a typical form of measles after the usual period of incubation.

Convalescent measles serum, plasma, or whole blood can be used to produce passive immunity if injected within the first four or five days after exposure, and to modify the character of the disease when injected after the fifth day.

The dose of serum during the first four days after exposure is 2.5 c.c., during the fifth and sixth days 5.0 c.c., and during the seventh and eighth days 7.5 c.c. Doses of from 2.5 to 3.0 c.c. from the fifth to the tenth day after exposure modify the attack. These doses are calculated for children 3 years of age.

HENN, Chicago.

RHEUMATIC HEART DISEASE IN CHILDREN UNDER TWO YEARS OF AGE. B. S. DENZER, J. A. M. A. **82**:1243 (April 19) 1924.

Denzer reports three cases of rheumatic fever with heart disease, in children under 2 years of age. It was found at necropsy that these children had either subcutaneous fibroid nodules or Aschoff bodies. The author raises the question whether the present view of rarity of rheumatic heart disease in infancy might not be revised.

THE PATHOGENESIS OF RHEUMATIC FEVER. HOMER F. SWIFT, J. Exper. Med. **39**:497, 1924.

The manifold clinical signs of rheumatic fever, and the occurrence of joint pain in so many diseases, have caused much confusion in our conception of the essential nature of the disease.

Nonhemolytic streptococci have been claimed to be the causative microorganism, but the failure to recover them from the majority of patients and to reproduce the classical disease by inoculation into animals leaves the question unsettled. For a better understanding of rheumatic fever we are forced to a continued bedside study and tissue examination.

Excrescences made up of elements of the circulating blood occur along the free margins of the heart valves, at places where the endothelium has disappeared. Evidences of proliferation are seen under the endocardium in the heart valve. Characteristic lesions occur in the mural subendocardial region without primary injury to the endothelium and in the base of the valve leaflets. It is not difficult to conceive of the primary injury of the valves occurring in their substance rather than on their surface. The possibility of swollen covering endothelium being broken by impacts against an opposing leaflet is easily seen.

Lesions similar to Aschoff bodies found in the diseased pericardium indicate that the essential or primary pathologic process is similar to that found elsewhere.

The most generally recognized specific histologic lesion of rheumatic fever is the Aschoff body which is a submiliary nodule located in the myocardium. Changes in the blood vessels are common. Interference with circulation must lead to disturbed nutrition of the muscle and impulse conducting fibers. Bedside and electrocardiographic investigation indicated that the myocardium or conduction system was disturbed in about 95 per cent. of cases.

The essential histologic picture of subcutaneous nodules is similar to that of the Aschoff body.

Fahr found changes in the capsule of the knee comparable to myocardial lesions. Coombs found similar changes in a shoulder joint. The capsule of the knee and ankle revealed lesions comparable with those of the heart and subcutaneous nodules. Numerous nerves in the joint capsule and surrounding ligaments easily explain the pain. The finding of lesions in the joint capsule of a patient from whom all clinical signs of arthritis had disappeared under neocinchophen, indicates that the essential rheumatic process may go on despite these antiphlogistic drugs.

Studies of the brain in chorea in the literature indicate that the chief lesion is vascular in origin. Thrombi, endothelial proliferation, and perivascular collections of round cells with small focal changes in the contiguous nervous tissue have been described. The usual peculiar response of the central nervous system to injury would naturally cause a different histologic picture here than elsewhere.

The finding of Aschoff bodies and subcutaneous nodules in chorea support the viewpoint that the lesions in the various organs are due to a common causative agent—the infective agent, or virus of rheumatic fever.

With this conception of the essential pathology of rheumatic fever, namely, disseminated focal submiliary nodules, with edema in the contiguous tissues during the acute stages, combined with lesions of the blood vessels, it is not difficult to reconcile the manifold and apparently unrelated manifestations of the disease.

McClure, Evanston, Ill.

Chronic Infections

The Recent Recrudescence of Epidemic Infantile Paralysis. Herman B. Sheffield, M. J. & Rec. **119**:72 (April 2) 1924.

The author calls attention to the fact that there has occurred in New York another epidemic of poliomyelitis of moderate severity. He does not give the number of cases reported to the health department, but states that "as usual, a far greater number remained unreported for fear of quarantine." The author has seen eighteen cases with no fatalities, which is characteristic of this epidemic. The initial symptoms were not characteristic, as a great

majority of the patients complained of severe pain in the muscles, while the paralysis did not appear until rather late (in two patients after ten days). Symptoms were attributed by many physicians to digestive disturbances, rheumatism or influenza. The somewhat detailed description of the pathologic anatomy should be seen in the original article. The author is opposed to the use of serum and apparently did not apply it in any of his cases.

BYFIELD, Chicago.

THE IMPORTANCE OF THE NEWER SEROLOGICAL METHODS IN CONGENITAL SYPHILIS. FRIEDRICH SZIRMAI, Jahrb. f. Kinderh. **104**:257, 1924.

The Wassermann reaction was found to be more sensitive than the Meinicke-Trübungs, Dold, and the Sachs-Georgi reactions, which showed degrees of sensitiveness in the order named. These precipitation reactions were therefore more definitely diagnostic of syphilis than the Wassermann. Practically all of the untreated cases which were clinically congenital syphilis showed a positive Wassermann reaction, and only a few of the treated cases were negative. A few non-specific positive reactions were obtained in scarlet fever, tuberculosis, chorea minor, and varicella, more often with the Wassermann reaction than with the precipitation reactions. The Meinicke-Trübungs reaction was positive in the mothers of syphilitic children more often than was the Wassermann reaction. The using of duplicate tests, therefore, showed some latent cases, and decreased the number of uncertain reactions.

SUTHERLAND, Chicago.

"LUES NERVOSA" AND THE INFLUENCE OF IDIO AND PARAKINETIC SYPHILIS ON PROGENY. I. HUSLER, Ztschr. f. Kinderh. **37**:200, 1924.

One hundred and thirty-two living children from fifty-two families, in which the mother or father or both had syphilis of the central nervous system, were studied. For comparison, the data from a series of 142 children whose parents (sixty) had visceral syphilis and from a series of 160 children of fifty-one nonsyphilitic parents were used. The results of pregnancies are given in the tables:

52 families with central nervous system syphilis—245 births.

Deaths	Per Cent.
Abortions	20.4
Premature births	3.7
Infants	17.9
Older children	4.1
Total	46.1
Living	53.9

60 families with visceral syphilis—276 births.

Deaths	Per Cent.
Abortions	26.4
Premature births	1.1
Infants	20.3
Older children	3.62
Total	51.5
Living	48.5

Over half of the children of the parents with syphilis of the central nervous system were not syphilitic, while only one-fourth of the offspring of the visceral syphilis group were absolutely free from syphilitic manifestations. Thirty-one per cent. of the children of the former group showed disturbances of the central nervous system, 30 per cent. of the latter. In the control group, no syphilis, 18.75 per cent. of the children had disturbances of the central nervous system.

PETERMAN, Rochester, Minn.

EXPERIMENTAL OBSERVATION ON THE "CURE" OF SYPHILIS IN THE RABBIT WITH ARSPHENAMIN. ALAN M. CHESNEY and JEROLD E. KEMP, J. Exper. Med. **3**: 553, 1924.

From the literature it would appear that there is no unanimity as to the possibility of curing syphilis in rabbits with arsphenamin. From experiments

in which the reinoculation method was used as a test it would seem impossible to eradicate the infection once it is established. With the method of lymph node transfer as a test, it seemed possible to produce a cure in rabbits. There is no unanimity as to whether or not a successful reinoculation of a rabbit with syphilitic virus indicates the cure of a preexisting infection.

The authors attempt to answer the questions as to whether syphilitic rabbits can be rendered free of the infection by administration of arsphenamin, as judged by lymph node transfer, and whether rabbits thus freed from infection are susceptible to reinoculation with the same strain of spirochetes. Twenty-two male rabbits were infected with *Spirocheta pallidum*. Ten received six weekly intravenous injections of 10 mg. per kilogram of arsphenamin, treatments being begun 127 days after inoculation. Twelve rabbits were left untreated. From thirty-two to thirty-eight days following the final injection of arsphenamin, both popliteal nodes were ground up and injected into the right testicle of each of two rabbits. The animals were observed for ninety days before being discarded as negative.

The lymph node transfers from the ten treated animals failed to produce syphilis in all cases. The nodes from the twelve untreated animals, produced positive results in ten cases.

Reinoculation was done in both treated and untreated animals forty days after completion of treatment, or 209 days after the original inoculation. Testicular emulsion containing motile spirochetes of the same strain was injected intradermally at the base of the right ear. Nine days later a drop of the testicular emulsion was rubbed into the scarified epidermis of the left ear. Reinoculation of ten treated and nine untreated rabbits failed to produce lesions at the site of inoculation.

These experiments show that the administration of arsphenamin as described is sufficient to eliminate an existing syphilitic infection in rabbts as determined by lymph node transfer, performed from thirty-two to thirty-eight days after discontinuing treatment, when the infection has been present 127 days before the treatment was begun. These results are in agreement with those of Nichols and Walker. Whether or not these animals continued to harbor spirochetes elsewhere in the body is still debatable. Concerning the reinoculation experiment the question arises as to whether the absence of local lesions at the site of inoculation is evidence that infection has not taken place. The authors are inclined to the view that the animals were in reality not reinfected. The failure of reinoculation in the treated animals could equally well be interpreted as evidence that the animals were immune, as that they were still infected. They consider the demonstration of the presence or absence of lymph node infection a better criterion for the evaluation of an antisyphilitic agent than the method of reinoculation as carried out in their experiments.

McCLURE, Evanston, Ill.

Internal Diseases

TWINS WITH HYPERTROPHIC PYLORIC STENOSIS. LESLIE H. MOORE, South. M. J. **17**:187, 1924.

This case report is quite unique, in that the condition developed simultaneously in both twins and ran quite a parallel course. Vomiting began in the second week. The Rammstedt operation was performed successfully on both patients at the end of the fourth week. Vomiting ceased, but death ensued in one infant forty-eight hours after operation, apparently from respiratory failure, and in the second child eight days after the operation, apparently from the same cause.

SCHLUTZ, Minneapolis.

CASE OF HENOCH'S PURPURA. DONALD KILGOUR, Canad. M. A. J. **14**:302 (April) 1924.

The author gives an excellent review of the present day knowledge of the subject. The classification of Minot and Lee is given as the most acceptable. Henoch's purpura is considered to be an idiopathic purpura other than hemorrhagic purpura, the latter term being limited to those cases of purpura showing a diminution in blood platelets.

Nothing new is brought out in pathology or treatment. The use of blood transfusion in the severe anemia accompanying the condition is stressed.

One case is reported in a boy, aged 5 years, who developed a very severe secondary anemia, with hemoglobin, 20 per cent., and a red count of 1,000,000. Transfusion with 300 c.c. of blood from his father, using direct paraffin method, resulted in a rapid cure. The unusual features in the case were severe epistaxis and the very severe secondary anemia.

MOORE, Omaha.

SUDDEN DEATH ASSOCIATED WITH THE INJECTION OF FOREIGN SUBSTANCES. R. W. LAMSON, J. A. M. A. **82**:1091 (April 5) 1924.

Lamson has tabulated the reported deaths following injection of foreign material since 1894. In one group are forty-one cases in which death may have been due directly or indirectly to the protein injected. In the other group of twenty-one cases death seemed due to primary disease or some other cause not associated with the injected substance. Diphtheria antitoxin was the protein injected in all but ten of the cases. This does not indicate an inherent toxic action of the diphtheria antitoxin itself but rather that this serum is used more frequently than any other serum. Fourteen cases gave a history of asthma or hay-fever. Previous injections had been given in eight cases and in thirty-one there was no history of injection. The treatment of asthma with diphtheria antitoxin or normal horse serum resulted in five deaths. The sex of the patient was not indicated in four cases, but in others twenty-six were males and eleven females. Most of the patients were under the age of puberty. In a majority of cases death follows in a short time. When the fatal reaction is delayed for a matter of hours, it is difficult to ascribe to the serum specific or nonspecific effect.

The diagnosis of status lymphaticus was made at necropsy in three cases. The relation of the thymus to the cause of death here, as in cases during anesthesia, is not settled. Unfortunately, in only a few cases was necropsy obtained, and in these there was no consistent pathologic finding.

A negative scratch test for hypersensitiveness is not a sufficient reason for a nonspecific reaction. In a few of the observed cases the amounts injected were so minute that it is difficult to believe that the nature of the injected substance could have very much to do with the fatal result. One is inclined to assume individual physiologic peculiarities on the part of the patient as the determining factor.

HENN, Chicago.

OLEIC ACID HEMOLYSIS IN INFANTILE ANEMIAS. KARL BENJAMIN, Jahrb. f. Kinderh. **104**:277, 1924.

Febrile infections and overfeeding with milk lead to anemia, but the mechanism of this is not well understood. Hemosiderosis in the liver and spleen, and increased excretion of urobilinogen in the stools point toward increased blood destruction rather than a deficiency in the food. The whole blood of anemic infants showed hemolysis with a lower concentration of sodium oleate solution than did the blood of normal infants. Cholesterin, which is a protective substance against hemolysis, is decreased in the anemias even

more than is the dried residue. Other substances, probably proteins of the serum, also neutralize the oleate. The author concludes that overfeeding with milk, and to a greater extent febrile infections, cause anemia by decreasing the cholesterin and the albumin of the serum, thus leading to increased hemolysis.

SUTHERLAND, Chicago.

CLINICAL EXPERIENCES IN ORGANOTHERAPY WITH SPECIAL REFERENCE TO THE STIMULATION OF BODY GROWTH. T. A. MCGRAW, Endocrinology, Bull. of the Association for the Study of Internal Secretions **196**:234, 1924.

The author presents a number of his clinical experiences with organotherapy, the patient in every case being a child suffering from retarded physical development. The patients are divided into two classes: (1) with definite symptoms of endocrine disease; and (2) without definite symptoms of endocrine disease. Thirteen cases are presented with their essential histories and graphic growth charts and photographic illustrations. There is a brief discussion of the cases, especially as regards rational of treatment instituted. The author gives several pertinent therapeutic suggestions. He deplores the all too prevalent practice of organotherapy in a hit-and-miss manner and stresses the importance of a careful diagnosis from the endocrine standpoint, supplemented by roentgen-ray and laboratory examinations. He condemns the prevalent use of pluriglandular mixtures, as sold by the mail order houses. He would institute treatment with the extract of the gland standing out as primarily affected, adding to this the extract of the other glands as indicated.

WADDELL, University, Va.

ABDOMINAL PAINS IN CHILDREN BEYOND THE AGE OF INFANCY. HARRY S. SNYDERMAN, M. J. & Rec. **119**:78 (April 2) 1924.

The author properly calls attention to the obscure etiology of colicky abdominal pain in childhood, excluding referred pains which are due to acute conditions. He assumes that the commonest cause is some form of indigestion, especially in those cases where a large amount of mucus is present in the bowel movement (Mucus Disease, Eustace Smith). Other causes which he includes are: irritation from undigested vegetable matter due to imperfect mastication. This latter sometimes is the result of defective teeth. Illustrative cases are quoted. Thread-worms and other intestinal parasites, constipation, cyclic albuminuria, renal calculus, movable kidney, tabes mesenterica or caseation of the mesenteric glands and biliary colic (gallstones). The case illustrating this latter condition in a child, aged 9 years, was confirmed by operation.

BYFIELD, Chicago.

Diseases of the Skin

A SURVEY OF ULCUS TROPICUM. CORPUS TEOFILO, J. A. M. A. **82**:1193 (April 12) 1924.

Tropical ulcers usually appear in cachectic persons or in barefooted school children during the wet months. They are thought to be caused by a spirochete described as Eggerts' Type A. The spirochetes may be seen in diluted carbol-fuchsin stains of oozings from edges of the ulcers. The disease is contagious and is often spread by school children in constant contact with one another. Treatment is varied but the most effective is arsphenamin (3 per cent. solution applied locally), and the next is Vincent's Powder, composed of one part sodium hypochlorite and nine parts boric acid.

HENN, Chicago.

Isolated Favus of the Eyelid. Report of a Case. Sigmund S. Greenbaum, Am. J. Ophth. **7**:6, 1924.

A boy, white, aged 10 years, developed a yellow, cupshaped crust on the lower lid just beneath the lashes. On removal, the underlying tissue was erythematous with epidermic infiltration and several small scattered erosions. The crust was made up of mycelium and spores. On culture the organism grown was *Achorian quinckeanum* (mouse favus).

Wescott, Chicago.

The Therapy of Constitutional Eczema in Infants. A. S. Sokolow, Jahrb. f. Kinderh. **104**:91, 1924.

A series of eleven infants with chronic eczema was injected parenterally with foreign protein in the form of milk, in doses of from 0.2 to 0.4 gm., at intervals of from two or four days. There was complete recovery in five cases and marked improvement in three cases, giving a good result in 73 per cent. The improvement in the eczema was definite after three or four injections. The chief reaction was a fever, at its maximum in seven hours. Only permanganate baths were used locally.

The writer suggests that even better results should be obtained if skin sensitization tests were made, and the injected protein varied, depending on the infant's sensitization.

Sutherland, Chicago.

Nervous Diseases

Sense Perception of the Prematurely Born. Albrecht Piper, Jahrb. f. Kinderh. **104**:195, 1924.

The effect of sound, light, pain and cold stimuli on the respiration of the sleeping infant was recorded on a kymograph tracing by means of a Marey tambour. The premature infants reacted as readily as the well developed newborn infants. The writer suggests this method to test sensation in idiots.

Sutherland, Chicago.

A Note on the Comparative Histopathology of Acute Anterior Poliomyelitis and Epidemic Encephalitis. George B. Hassin, Arch. Neurol. & Psychiat. **11**:28 (Jan.) 1924.

Hassin, in his studies of a case of epidemic encephalitis and one of anterior poliomyelitis, concludes that, although grossly, the two conditions appear very similar; they have a distinctive histopathology as well as distinct clinical and serologic characteristics. He states that in both conditions there may be widespread changes throughout the brain and spinal cord and that these changes in the medulla, pons and other parts of the brain cannot be differentiated from each other in all cases. Differentiation, however, is possible by a study of the changes in the spinal cord. There the inflammatory changes are usually much more marked in anterior poliomyelitis, in which the smaller capillaries are involved. However, the distinguishing point is the fact that the degenerative changes in the motor neurons are far greater in anterior poliomyelitis than in epidemic encephalitis. Also that in the former these changes are confined to the motor neurons, as is not the case in the latter. In anterior poliomyelitis the degenerative and inflammatory changes decrease in severity in the medulla, pons and cortex, while in epidemic encephalitis the changes are most marked in the brain, and decrease in severity in the spinal cord. There were also found in the central canal of each cord hemotogenous elements resembling those present in the perivascular spaces and in the parenchyma. This, the author points out, may be interpreted as a proof that the flow of the contents of the perivascular spaces is not only toward the subarachnoid space but also toward the central canal.

FAMILIAL MUSCULAR DYSTROPHY. H. P. MILLS, THOMAS H. HAINES and MINA A. SESSIONS, Arch. Neurol. & Psychiat. **11**:43 (Jan.) 1924.

Mills, Haines and Sessions report a series of six cases of muscular dystrophy occurring in one family. Five were boys and the other a girl. All but one boy, who died of pneumonia at the age of 19, are living. The ages of the remaining children range from 9 to 29 years. The boys all suffer from primary type of atrophy, affecting the shoulders and arms or trunk and thighs. The girl suffers from the pseudohypertrophic form and is the eldest of the children affected. The facial muscles were unaffected in all the patients. The condition appeared in all the children at the age of 7 years. In one of the boys, a contracture was present in the hip joint but was not found in any of the others. Examination of the blood and spinal fluid revealed no abnormalities. The blood sugar was found to be within normal limits. Basal metabolic rates were taken on the five living children and ranged from a minus 22 to a plus 10. No abnormalities were brought out by roentgen-ray examination of the skull. Vasomotor disturbances were present in practically all cases. The authors point out that the one case of the pseudohypertrophic type, occurring in the eldest girl, does not conform to the characteristics reported by Gordon, who stated that such cases usually die young, were more frequent in boys and are apt to present no history of other cases in the family. There were five other children in the family who were healthy, and no such trouble was present in the eleven grandchildren, ranging from the age of 1 to 11 years.

AVERY, Chicago.

Diseases of the Eye

ETIOLOGY OF KERATOMALACIA. EUGEN STRANSKY, Jahrb. f. Kinderh. **104**:183, 1924.

The writer presents two cases of keratomalacia which were typical avitaminosis and hypovitaminosis. The former patient improved rapidly on cod liver oil and butter, though the latter, a patient with pyloric stenosis with marasmus, died. Other cases which developed in the course of other conditions, such as dysentery (Flexner type), severe chronic dyspepsia, and acute dyspepsia could not be ascribed to a lack of vitamin A in the diet. These patients all died. The conclusion is that xerophthalmia is dependent on a lowered resistance of the whole organism and especially of the cornea to infection of any kind. This lowered resistance may be due to avitaminosis or hypovitaminosis or to enteral or parenteral infections. In the latter cases vitamin A is without effect on the xerophthalmia.

SUTHERLAND, Chicago.

HEREDITARY CATARACT. ALBERT L. BROWN, Am. J. Ophth. **7**:33, 1924.

Three generations of descendants of one family have been under observation since 1911. Of eight descendants of the first patient seven have congenital cataracts. In each case the transmission was from a female, but both sons and daughters were affected. The husbands had normal eyes and there was no blood relationship between husband and wife. Nothing new is added by the author as to whether the defects are developmental or metabolic, but he does stress the sociologic question of marriage among such persons and reports that the answers to questionnaires sent to ophthalmologists and superintendents of homes for the blind showed the majority in favor of limitation of marriage.

WESCOTT, Chicago.

Surgery and Orthopedics

CORRECTION OF STRUCTURAL LATERAL CURVATURE OF THE SPINE. R. W. LOVETT and A. H. BREWSTER, J. A. M. A. **82**:1115 (April 5) 1924.

The authors are using in treatment of organic or structural scoliosis, a new jacket that will comfortably correct and at the same time permit of mobilization of the spine.

SILENT FOCI OF LOCALIZED OSTEOMYELITIS. D. B. PHEMISTER, J. A. M. A. **82**: 1311 (April 26) 1924.

In four cases reported, Phemister found osteomyelitis lesions which had proceeded to the stage of complete destruction of the bone area involved, with surrounding new bone formation before there were any local symptoms whatsoever. The only places where symptomless osteomyelitis has been thought to develop were pockets about the roots of teeth. These are not thought to be related to the silent foci which usually arise as complications or sequelae of diffuse staphylococcus osteomyelitis. Phemister suggests that sometimes a routine search by palpation and roentgenography is indicated, and that these silent foci may explain localized symptomless areas of bone sclerosis.

HENN, Chicago.

A CASE OF MASSIVE HYPERTROPHY OF THE BREASTS IN A YOUNG GIRL. THEW WRIGHT, Bull. Buffalo Gen. Hosp. **2**:31, 1924.

Wright reports a typical case of puberty hypertrophy of the breasts, and calls attention to the fact that it may be due to an endocrine imbalance, whereby the normal inhibitory influence to mammary hypertrophy fails to act at the proper time.

BACON, Chicago.

CONGENITAL CYSTIC DILATATION OF THE COMMON BILE DUCT. G. L. MCWHORTER, Arch. Surg. **8**:604 (March) 1924.

The author operated on a woman, aged 49, who had been suffering from attacks of pain in the right upper abdomen associated with jaundice since she was a baby. She had previously had a drainage of the gallbladder performed. His findings at operation were extremely interesting. The gallbladder was small and distinctly differentiated from a dilated cystic duct by a narrow constricting sphincter of scar tissue and muscle. The cystic duct opened into an enormous balloon-like common duct, that apparently had no communication with the duodenum which could be demonstrated. This cavity contained several small cholesterin stones as did the greatly dilated hepatic ducts. The dilated common and cystic ducts were removed and an anastomosis made between the duodenum and hepatic duct. The patient made an excellent recovery, in spite of a temporary duodenal fistula.

The literature contains the records of but forty-seven similar cases, none of which was diagnosed before the abdomen was opened. The condition is a progressive one, and a good prognosis is dependent on early operation and effective internal drainage of the bile. Of this series, including the author's case, only nine were successfully operated on and permanently cured.

In view of the difficulty of the preoperative diagnosis, we believe that the recent experimental work of Evarts Graham on detection of abnormalties of the gallbladder by means of a dye excreted through the bile and casting a shadow in the roentgen-ray picture would materially aid in such a case as this one.

CONGENITAL DEFICIENCY OF THE DIAPHRAGM. E. C. CUTLER and S. F. COOPER, Arch. Surg. **8**:506 (March) 1924.

There are three classes of congenital lesions of the diaphragm (1) true hernia with a sac covering the displaced intestine; (2) false hernia, or hernia without a sac; and (3) eventration. The diaphragm develops from four different sources: Its ventral pericardial portion is formed from the septum transversum, its lateral portion from the pleuroperitoneal membranes and the medial dorsal portion from the primitive mesentery. Its muscular portion comes from a pair of premuscle masses which lie on each side of the fifth cervical somite. This muscle goes caudad with the septum transversum, carrying the phrenic nerve with it, and develops chiefly in the dorsal portion of the diaphragm. According to Hume, the types of congenital diaphragmatic hernia may be classified as arising as follows:

1. Through the hiatus pleuroperitonealis. This is the commonest type and is probably due to the intestine following the lung bud as it leaves the abdominal cavity (always false).

2. Through the dome of the diaphragm. This may be true or false and may be caused by a weak point in the diaphragm. These cases are often associated with incomplete rotation of the intestine.

3. Through the esophageal orifice. The stomach may fail to migrate below the diaphragm or may migrate late, leaving a large esophageal opening.

4. The entire left half of the diaphragm may be absent, a very rare condition. The fact that congenital deficiency of the diaphragm occurs much more frequently on the left than the right may be explained by the distribution of the vascular elements which play such an important part in the early formation of the embryo. The vascular concentration in the region of the right side of the diaphragm would contribute to the strengthening and blocking of the pleuroperitoneal canal, while atrophy of the common cardinal vein on the left would contribute to the weakening of the left half of the diaphragm.

The authors in operating on an 8 weeks' old puppy, observed that the animal had great difficulty in attempting to vomit and finally died on the table. At necropsy a congenital opening was found in the anterior part of the right diaphragm and through this opening had passed the fundus of the stomach, the spleen, part of the great omentum, about two thirds of the liver and the gallbladder.

In children, the symptoms of such a lesion are usually due to herniation through a relatively large opening of a considerable part of the abdominal gastro-intestinal tract, occurring as a result of straining or changing position, giving rise to an acute dyspnea with asphyxia. Examination should reveal unusual tympanitis in the chest and obscure breath sounds. If the opening is small, a loop of bowel may be kinked and give rise to signs of obstruction. In the majority of cases, however, there are no unusual symptoms at all or merely indigestion. Roentgen-ray examination should make certain the diagnosis and differentiate between the types. Treatment must be surgical if the severity of the symptoms warrant any interference at all. The thoracic route is the one of choice. By excision of the sixth, seventh or eighth ribs posteriorly and laterally, an excellent exposure of the diaphragm is obtained.

MILLER, Chicago.

UNRECOGNIZED FRACTURES OF THE SKULL IN INFANTS AND CHILDREN. ALFRED W. POLLAK, M. J. & Rec. **119**:65 (April 2) 1924.

This is a clinical article giving six case histories of traumatic fracture of the skull in children over 3 years of age. Three roentgen-ray pictures are shown. The author classifies the future effects as follows: (1) most fractures without depressions become absolutely well; (2) cases of mild con-

cussion with loss of consciousness for a few seconds often show subsequent changes in disposition; (3) chronic headaches, usually of a constant dull aching type; (4) inability to stand high temperatures; (5) epilepsy, due to irritation, produced by bony spicules from depressed internal table, with blood effusion, resulting in thickening of the meninges and the formation of cysts, either beneath the site of the injury or on the opposite side.

He concluded by stating that many of the obscure brain conditions of later life may have their origin in a mild degree of concussion and suggests that every case where a child has fallen or been struck upon the head, a roentgen-ray examination of the skull be made and careful history taken in order that nothing be overlooked.

APPENDICITIS AND PERITONITIS COMPLICATING SCARLET FEVER. BENJAMIN NEWMAN, M. J. & Rec. **119**:80 (April 2) 1924.

The author believes that scarlet fever is rarely followed by acute appendicitis or peritonitis developing during the eruptive stage or during desquamation. After referring to ten articles in the literature, he gives the experience of a case of his own observation. The patient, aged 6 years, was suffering from typical scarlet fever. On the fifth day the temperature rose to 102 F. and abdominal pain complained of. Operation was performed and localized appendicular abscess discovered. The recovery was without complication.

BYFIELD, Chicago.

Miscellaneous

POSTDIPHTHERITIC PARALYSIS. ALAN G. TERRELL and DAVID H. HALLOCK, M. J. & Rec. **119**:68 (April 2) 1924.

This is a clinical article, with two case reports, one of a child aged 3½ and the other 21 months, both of which died. The first patient was given 3,000 units of diphtheria antitoxin. Paralysis appeared one month after the onset of the disease at which time the child was admitted to the hospital, dying a few days afterward. In the second case, 20,000 units of antitoxin were given. Paralysis followed five weeks later. The first patient died of respiratory paralysis and the second, of paralysis of the heart. The authors urge the preliminary giving of antitoxin before the culture report has been returned. They are also impressed with the importance of passively immunizing all contacts. They might well have called attention to the importance of prophylaxis through active immunization with toxin-antitoxin.

JUVENILE TABES DORSALIS WITH DESTRUCTION OF HIPS FROM CHARCOT'S DISEASE. WALTER G. ELMER, M. J. & Rec. **119**:70 (April 2) 1924.

A case report of Charcot's disease, developing in a girl aged 14 years, who, while doing gymnastic exercises, was thought to have dislocated her right hip, is presented by this author. When surgically treated by an orthopedic surgeon, a diagnosis of syphilis was made and treatment with neoarsphenamin begun. At that time the knee reflexes were found to be absent and the pupils did not react to light. When examined four years later, the same findings, together with Romberg's sign, were also noted. Blood Wassermann was negative with the spinal fluid strongly positive. Four years afterward, both tests were negative.

LONDON LETTER. M. J. & Rec. **119**:370 (April 2) 1924.

The recent epidemic of influenza in Great Britain has led to a marked increase in the infantile death rate. Whooping cough was also partly responsible for this increase.

RELATIONSHIP OF INFANT AND CRUDE MORTALITY RATES. DWIGHT M. LEWIS, M. J. & Rec. **119**:76 (April 2) 1924.

The author's conclusions appear in his summary, which reads thus: "yearly variations of infant mortality rates in cities where so-called standard measures for its reduction have been effective for years without decreasing such variations, statistically as well as epidemiologically, would seem to be influenced by the respiratory factor of the crude mortality rate."

THE CHILD FAMILY RELATION: A PSYCHOSEXUAL STUDY. J. F. W. MEAGHER, M. J. & Rec. **119**:88 (April 16) 1924.

This should be read in the original. The following sentences are taken at random.

Compensatory traits cover feelings of inferiority or of insufficiency. Compensations are to hide tendencies of an opposite nature; this being a self defense mechanism. Most normal people feel the need of developing self respect and of acquiring social esteem.

A person may inhibit the ideas of sex but not the energy.

Sex undergoes marked depression in all children from infancy, but is greater in girls than in boys . . . Without doubt, the most important lesson to learn in child psychology is to bring up the child so that at the end of adolescence it will effectively emancipate itself from the family. . . The acts of the family have great social value. . . Nervous types very readily absorb the home difficulties. Family feuds are often very intensive and constitute a great hindrance to the nervous patient in such a family. It is well to remember that there are two extreme ways of training a child. One is that of excessive love and coddling and the other is with a lack or absence of love and too great severity. . . It is really a bad mistake on the part of the parents to try to inculcate ideas that everything relating to sex is shameful or degrading. . . Certainly it will be admitted that the absence of happiness in a nervous individual is clinically of more importance than the presence of a dermatographia or an indicanuria.

CHILD WELFARE. M. J. & Rec. **119**:367 (April 2) 1924.

The following recommendations were made:

Infant rearing centers: It was recommended that, because of the difficulty of supervising the care of infants placed out promiscuously, rearing centers be established under the direction of qualified medical officers.

Improvement of day nurseries: (1) All crèches should be under the direction of a physician; (2) separate rooms should be used for the cradles, the linen, disinfecting purposes and isolation of patients; (3) they should be open to the pupils of puericulture schools.

School hygiene: Close cooperation should exist among the teacher, the school physician and the school nurse. The teacher should be acquainted with the fundamental principles of hygiene. Each child should be submitted to an annual examination, of which a record should be kept, and proper action taken as indicated by these examinations. The attention of the public authorities was called from the point of view of prophylaxis, to the importance of the medical examination from the point of view of the teacher and to the value of dental hygiene.

BYFIELD, Chicago.

Book Review

SELECTED ESSAYS ON ORTHOPAEDIC SURGERY. By NEWTON M. SHAFFER. New York, G. P. Putnam's Sons, 1923. $5.00.

This volume, comprising essays on orthopedic surgery by Dr. Newton M. Shaffer, is typical of the writings of his day. They were mainly published in the late seventies and eighties of the nineteenth century.

It is particularly interesting to read the essays on chronic joint diseases, dealing with hip joint, ankle and spine. Shaffer's contention that conservatism is to be preferred to radicalism, is borne out by the present day teaching. At the time of his writings there was a wave of radicalism, in the treatment of tuberculous joints, sweeping the surgical world, particularly in hip joint disease, where excision was being widely advocated. Shaffer took the stand and proved that his results were better by conservative means than by operative procedures. The old controversy in the treatment of the hip joint diseases was then waging, that is, fixation versus extension, and the author's stand here again is borne out by subsequent experience, that is, that extension particularly in the acute and subacute stages is essential.

His teaching, that tuberculous abscesses should not be incised and drained, also has stood the test of time. It must be remembered that the tubercle bacilli had not been identified at the time many of these essays were written, and the disease was called strumous, scrofulous, and so forth.

Dr. Shaffer was a pioneer in the orthopedic work in America, and it is owing to the efforts of men of his type that the specialty today stands on the firm basis that it does.

The close clinical observations that these men carried on, so well evidenced in Dr. Shaffer's essays, might well be emulated today. Too great dependence on laboratory findings tends to weaken the clinical acumen of the modern medical man.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, Cook County Hospital, Nov. 20, 1923

H. W. CHENEY, M.D., *President*

CONGENITAL SYPHILIS. Presented by DR. ARTHUR W. STILLIANS (by invitation).

The first patient, a child, aged 2 years, illustrated symptomless syphilis. The child was brought in with condylomas but is now free except for slight staining where the condylomas were. The shape of the head and the carious upper incisors and first molars were suggestive. The feet were underdeveloped, and the child was mentally backward.

The second patient, a colored child, aged 5 months, had an enlarged epitrochlear gland and a maculopapular eruption around the anus, as well as a few inflammatory lesions which were not syphilitic. The principal features were the stains of the papular pigmented syphilid, which occur in both the white and colored races, and leave pigment for a long time. The child's hair looked as if it were worn off, the only kind of alopecia described in congenital syphilis, and Dr. Stillians thought it was due to rubbing rather than to the disease. A prolific growth of hair has been described as a symptom of syphilis, the so-called "syphilitic mop," but Dr. Stillians said he had never seen one.

The third case was in a boy, aged 7 years, who had a scar and also a recent gumma on the forehead. His teeth were in bad condition, the two central teeth sawedged in the manner typical of congenital syphilis. The upper front teeth were all mixed up and it was difficult to differentiate as to incisors and canines, none of them were notched, however. The gum was shrunken and the roots were exposed on one side. The molars were carious on one side both above and below. There were swellings of both the radius and ulna, due to periostitis. The lesions were bilateral and symmetrical in many places. A typical saber tibia and an enlarged epitrochlear gland were present on one side. Glandular swelling is not as reliable in congenital syphilis as in the acquired type. This patient was a brother of the first patient shown.

Dr. Stillians was interested in the differential diagnosis between the lesions of congenital syphilis and ringworm. The circinate syphilids in children usually have a narrow border of considerable height, smooth, and ranging in color from that of the skin to dark brown. The lesions of ringworm usually show vesicles or scales on a wider, less sharply elevated border.

The diagnosis between pemphigus neonatorum and bullous impetigo often comes up and is of great importance. The pemphigus lesions are usually found on the palms and soles and adjacent areas on the arms and legs, while the impetigo begins wherever the infection takes place and spreads asymmetrically over the body. Those Dr. Stillians had seen in syphilis were vesicles which were fairly thick walled. In bullous impetigo the lesions are very superficial, rupture very early and are usually larger than those of syphilis.

He was especially impressed some months ago with the difficulty of making a prognosis in congenital syphilis. The textbook rule is that the prognosis is worse according to the time and amount of eruption—the earlier and more marked the eruption, the worse the prognosis. In two cases observed at the same time in the hospital one presented a bullous syphilid which extended not

only over the arms and legs, but over the trunk as well. It was the most marked case he had ever seen and came on soon after birth. The eruption in the other baby came on three or four weeks after birth with a very scanty distribution of papules. They expected the first baby to die and the second one to get well but the reverse happened, and he was more guarded than ever in regard to prognosis in these cases.

COMMON FOOT DEFORMITIES IN CHILDREN. Presented by DR. BEVERIDGE H. MOORE.

In thinking over this subject it seemed to me that a very easy way to treat it was to divide the deformities into three classes: First, the deformities that are noticed before the child walks; secondly, those that are noticed after the child walks and, third the deformities of the feet, which appear in late childhood or early youth.

The most noticeable deformity in the early period before the child begins to walk is the congenital clubfoot, of which the equinovarus is the most common. It may be either bilateral or unilateral and there is no trouble in the diagnosis at birth. The only thing to look out for is to be sure that the condition is not due to spina bifida, because those cases give a different prognosis from the ordinary clubfoot.

As to treatment, the best plan is to start stretching and massage as soon as possible after birth. The mother can carry on this treatment until the foot is stretched and loosened, during the first month or six weeks, and then it is not nearly so hard to cure later on. When the child is about 6 weeks old I like to begin putting on casts, a plaster bandage being put on to correct the foot and twist it around gradually to a normal position so that it will be held and the bones will gradually grow in the position they should be. By doing this early one can often save the child an operation, or if an operation is necessary a simple tenotomy can be done later.

The valgus deformities are not so common but are occasionally seen. They are usually associated with deformity of the tibia or absence of the fibula.

Another deformity that is noticed early is the presence of supernumerary toes, for which the only treatment is removal. It seems to me it is well to wait for this until the child is gaining well, and in good condition generally, that is, some time during the first year because the child has not yet learned to walk and there is much less trouble in learning then.

The deformities noticed before the child begins to walk are nearly all gross deformities. We sometimes see flatfoot before the child walks but it is not of much significance. Most children have a low arch with a large pad of fat, and there is not much difference in the shape of the child's foot then.

Taking up the deformities that are noted after the child begins to walk, they are much more frequent, but not so marked. The first is weak ankles. The inner malleolus may almost touch the floor and the whole foot will be rolled outward. Those conditions can best be treated in little children by having the shoe built up on the inner side so that the weight is thrown to the outside of the foot, thus giving the ligaments and muscles on the inner side a chance to shrink and take up their work. It is difficult to get a child of this age to take regular exercise, and the easiest way to treat them is to have laced shoes with heavier soles than usual and have them built up so that the weight is thrown to the outer side. The mothers invariably ask about the so-called "weak ankle" shoes but I never thought they did any good.

Another thing the mothers notice is that the child toes in. This is really rather a good fault because if the child walks pigeon-toed it means that he is getting a good gripping action of the toes and this usually results in a good, sound pair of feet later on. Unless it is too marked and the child gets one foot in front of the other, it is hardly necessary to do anything for it. If necessary, one can have the shoe built up on the outer front part of the sole

so that that part comes in contact with the ground first, thus twisting the foot out. Turning the foot out, or everting it is worse because it throws more strain on the ankle. It can be much more easily corrected than the other condition by having the heel built up on the inner side as well as on the sole, tending to throw the foot inward.

Taking up the deformities appearing in late childhood or early youth, we then see the flatfoot when the patients are about 11 or 12 years old. We often get a history of a child growing very rapidly for a time, and then the mother begins to notice that the feet are weak and the arches come down. This is a matter of the child outgrowing its strength, and the muscles of the feet not keeping pace with the rest of the body. It is a good plan to use foot plates or metal arches, because you want to strengthen the arches and make them do the work. I believe the best treatment is to have a child wear a fairly wide shoe, well fitted, with a good shank. The principal thing is to get the muscles of the leg and arch built up so that they will support the foot and then it will be permanently well. That can be done by developing the muscles on the bottom of the foot and the tibialis anticus and tibialis posticus.

The cases should be classified, and exercise must be carefully graded and planned with considerable skill to have them done right. It is also a good plan to build up the shoe so that the foot will turn inward, throwing the weight slightly on the outer side.

Another deformity seen quite frequently about this time is hollow foot, feet that are contracted so that the weight bearing comes on the ball of the foot and on the heel. In these cases the toes are often retracted to a greater or less extent. Those cases come in different groups and need considerable support from a good, well fitted shoe that fits up under the arch. A good, wide heel of considerable height is necessary that will raise the heel well off the floor. Sandals for feet like this lead to a great deal of trouble. This hollow foot is one of the worst deformities that is seen although people very frequently are proud of the high arch.

Another thing that causes deformity of the feet is infantile paralysis. In all such cases deformity of the feet is due to unbalanced action, the stronger muscle outpulling the weaker. The main thing to do is to build up the weak muscle by exercise but this must be carefully graded. It does not do to tell the parents to have the child exercise and the foot will come out all right, for walking in these cases is very often very harmful and the child should have carefully supervised exercise as well as properly fitting braces to prevent the development of deformity.

A Case of Cleft Palate and Harelip Successfully Repaired. Presented by Dr. Herbert A. Potts.

Dr. Potts believes that first and last these cleft palate babies belong to the pediatrician. In many instances it would be criminal to try to repair a cleft palate in these tiny little things. (Pictures were here shown of emaciated infant.) This child came in three weeks ago in a worse condition than at present and has been under the care of a pediatrician since then. It is now in fair condition and will be checked up until the child is on a straight road to health. The nutrition is poor, the circulation is poor and any operation is likely to be a failure. Such cases belong to the pediatrician before and after operation.

As death at times occurs on the operating table, and some patients develop a high fever after operation, we should always be suspicious of a "lymphatic state." We can demonstrate a considerably enlarged thymus by means of the skiagraph and this precaution should be taken in all infants. If an enlarged thymus is found, operation should be deferred for some weeks or months during which time the enlarged gland can be reduced by means of roentgen-ray therapy.

In cleft palate and harelip I believe that the anatomy of the soft parts is practically complete in most cases and that the cleft is simply the result of the nonunion. The bony vault of the cleft palate is high, and the sum of the soft tissue is usually sufficient when dropped down to bring it together in the midline. The different types result from failure of union of the different portions. I saw recently a simple harelip with a split uvula, the palate and alveolar arch were complete. We frequently see just the split uvula. Then there is the double cleft palate in which the vomer is hanging loose in the vault, and the single one in which the right side may be perfect while on the left side the floor of the nose is absent. We may have a simple notch in the alveolar border or a simple notch in the lip. The majority of these deformities occur on the left side. In the double complete we have the double cleft of the lip in which the premaxilla is swung away out on the nose.

The cleft palate babies are not infrequently deficient in many ways. A certain number are idiots, and it seems the number of mental derangements is greater than in the otherwise normal babies. This picture (showing photograph) is that of an imbecile with a double cleft palate and harelip. The parents were anxious to have the appearance improved, so we repaired the lip but did nothing with the palate. There are not infrequently deformities of other parts of the body, a spina bifida, polydactylism, horseshoe kidney, absence of some of the toes or fingers or absence of the forearm or leg not due to pressure amputation by the cord.

This picture (indicating) shows a single cleft palate of the left side with an almost total absence of the forearm. This child has a lip cleft in the midline, a truncate nose, absence of vomer, meningocele in the vault of the pharynx, cleft palate, divergent squint and undoubtedly is a Mongolian idiot. (Showed series of pictures of different types of deformities.)

Of course, primarily, it is the care of these patients and not especially the repair of the deformities which should interest us. That means the children should be in good physical condition and gaining in weight. I think they are all pediatric problems. There is one common type of cleft palate and harelip in which there is a split in the arch combined with the harelip, which deserves special mention. These babies should have adhesive plaster strips placed across from in front of the ear on the well side; that is, on the long arm of the arch to in front of the other ear. The arch is usually swung out and there is protuberance of the lip on the long side. If the cleft in the lip is brought together, the plaster being cut out to accommodate the lip and nose, and the plaster is put on taut and kept tight daily, there will be a very much smaller cleft in the palate when one wishes to operate, and furthermore, the protuberant long arch will be pressed back into place. Excoriation of the skin can be avoided by cutting out the central portion and by restrapping it, putting the new plaster on top of the old.

As to the time to operate, in both cleft palate and harelip the ideal time is right away, in the first place on account of less scarring of the lip, and second because if the deformity is allowed to go on the high arch gradually flattens out. There is less space between the soft palate and the posterior wall of the pharynx in the infant than in the child 2 years old. Consequently, if perfect speech is to be attained it is necessary for the soft palate to close off the nasopharynx. If we wait until the child has grown, that space is greater and it is more difficult to restore the function of the palate. So it seems the best time to operate is the earliest, within a day or two or three for the lip, alveolar arch and nose, which we prefer to repair first, and then repair the hard and soft palate. At times we see that the mucoperiosteum is very thin and anemic. In these cases we prefer to make preliminary delayed flap operation, dissect it up from the bone and do nothing else for two or three weeks. By that time the flap is vascularized and the chances for good repair are better.

The repair of the nose is the most difficult. We have to make a floor for the nose and consequently we use all the mucoperiosteum we can. The majority

of cleft palates repaired by the sliding mucoperiosteal flap result in a new bony palate. There are enough osteoblasts sticking to the periosteum to form a new bridge underneath. There are three general types of repair. Dr. Brophy brings the maxillary bones together before the third or fourth month when the bones are pliable, and holds them there by means of wires passed through the bones and twisted over lead plates. Another type proposed by Lane is one in which the mucoperiosteum is dissected up and reflected across the cleft leaving a raw surface exposed. The operation of choice is that of von Langenbeck. The soft palate must be cut away from the posterior border of the hard palate in many instances in order to close the cleft. The one thing to be especially feared is shortening of the palate by scar contraction, as any raw tissue in the mouth will heal over by scar formation. On the nasal surface there is a denuded space which results in the shortening of the palate and this we always wish to avoid. We try in a number of different ways but whether we will succeed or not is problematical. As it is, we are careful to put some sutures on the nasal side of the soft palate in order to bring the mucous membrane in apposition, and leave as little denuded surface as possible.

The after care of the lip is very important and these edges must be kept absolutely sterile. Little scars that we pay no attention to on other parts of the body look bad on the face and we should always try to avoid them. Horsehair is the most suitable suture material. It produces no pressure necrosis and if the little serum that oozes out is kept away there is no scar. The food should be sterile, and if it does not cause too much disturbance the parts should be irrigated by the douche can or syringe.

At times the babies do not eat and it is desirable to feed them by gavage. Passage of the nasal tube is not difficult but passage of the tube by mouth may result disastrously.

Speech defect is important and much depends on the parents in teaching the child to speak slowly and distinctly, especially the consonants. One of the best ways to accomplish this is to teach the child or adult a foreign language. It is very difficult to correct the speech defect by repairing the palate of the adult, as the nasopharyngeal space has increased with the growth and as a rule better speech can be attained by means of an obturator. (A stereoscopic picture of a cleft palate and harelip was shown, with lack of union of the columella nasi and lip.)

CLINICAL CASES. Presented by DR. H. E. IRISH.

CASE 1.—A colored boy, presented because he showed a combination of things that are rather unusual. He had a neoplasm on the chest wall which contained pus, and which had been in existence for some time as a chronic abscess. It was attached to the sternum, which seemed to be thickened at the edge and then cupped out. The abscess had existed for about two months. There was pathologic change within the thoracic cavity as well as without, there being flatness in the region upward and outward from this area. No breath or voice sounds were transmitted. The area of flatness extended practically over the upper portion of the chest, the lower portion being approximately normal. Dr. Irish thought there was probably a circumscribed mass in the chest cavity as well as without. The roentgenograms verified the clinical findings.

The Wassermann and Pirquet reactions were negative. Aspiration of the chest wall gave a serous, very flaky and thick fluid. This was negative for bacteria and had been inoculated into animals. The insidious onset of the trouble and absence of cough or pain had made the condition a little puzzling. There was no history of a previous acute infection. The blood count showed 6,000 white blood cells, 55 per cent. small mononuclears, and 42 per cent. polymorphonuclears.

The question was whether the case was one of tuberculosis, syphilis, actinomycosis or a circumscribed empyema pointing at the location of the abscess. They were not yet sure as to the diagnosis, but thought that it was probably a case of tuberculosis, without positive confirmation in all directions. The patient was not sick, he was taking plenty of nourishment and his appetite was good.

CASE 2.—Laryngeal stenosis. A white child, the interesting features of whose case were the many reasons for the dyspnea. The patient had been in the hospital several times because of acute dyspnea and had shown many other conditions, among them a chest picture which corresponded with the shadows that are associated with enlarged thymus. He had also had a positive Klebs-Loeffler bacillus smear from the throat. His case had at one time been diagnosed as active diphtheria, and at other times he was considered a diphtheria carrier. In addition to that, during his long stay in the hospital he was suspected of having syphilis, because of condylomas. The dark field showed spirochetes and the Wassermann reaction was four plus. The Klebs-Loeffler bacillus organisms had been cleared up and the patient had improved greatly on active mercurial treatment, but apparently the thymus persisted. Direct laryngoscopic examination showed a thickening of the laryngeal wall posteriorly, but there was no other demonstrable lesion at that time. It was a question as to whether there was a syphilitic laryngitis plus an enlarged thymus, as well as at one time a laryngeal diphtheria. At the time of presentation the child was very hoarse and more out of breath than usual, due to an acute upper respiratory infection.

VENTRICULOGRAPHY. Presented by DR. GEORGE DAVENPORT.

Ventriculography was first introduced by W. E. Dandy in 1918, and to him is due not only the credit but to a large extent the clinical application thereof. More recently he has changed the terminology from ventriculography to cerebral pneumography. This term is more suitable and broader in its meaning, because often we not only see the outline of the ventricles on the plate, but the various passageways for the cerebrospinal fluid. Up to the present time we have done forty-five of these procedures.

Ventriculography, or cerebral pneumography, as we have practiced it is the replacement of the cerebrospinal fluid with air, in order to make the outlines of the ventricles and the passageways of this fluid distinctly visible on the roentgen plate.

The introduction of air can be accomplished by the intraventricular method, and when the fontanels are ossified we resort to a trephine opening in the skull, while in the infant we go through the open fontanel. The other method is by way of the intraspinal introduction of air. We have utilized both methods as indicated, always avoiding the intraspinal method whenever we felt there existed a tumor in the posterior fossa. In the intraventricular injection of air we always enter at a point anteriorly, which corresponds to Kocher's point of election. Entering at this point, the needle goes through the frontal lobe and enters the ventricle at the junction of the body with the anterior horn. After the obturator is withdrawn from the needle we attach a syringe with a two-way petcock. The fluid is withdrawn gradually, 5, 10 or 15 c.c. at a time, replacing the same by atmospheric air before more fluid is removed. This is repeated until we have removed as much fluid as possible. The patient lies with the neck flexed, face down and looking to the left.

The same procedure is followed in the intraspinal injection of air, except that the patient lies on the side with the head of the table tilted up at an angle of about 15 degrees. As soon as the injection is completed the pictures are taken, this is especially important when only a small quantity of air is introduced, so as to obtain pneumograms before absorption or dispersion of the air takes place.

We take four views as a rule, two lateral, one anteroposterior and one postero-anterior view. In the lateral views the saggital plane is kept parallel to the plate, while in the other views the saggital plane is kept at right angles to it. In all the work we have always maintained a definite distance from the plate to the target. In doing this we believe we can arrive at as nearly an accurate idea of the size of the ventricles as is possible in a human being, taking into consideration the numerous variations.

The time of exposure is the same as for any head picture, bearing in mind the fact that the infant's skull is more easily penetrated.

We have experienced what we have termed a compulsory gradual emptying of the ventricles brought about by a distortion or narrowing of the foramen of Monro on one side, so the fluid flows very slowly from the opposite side to the ventricle entered by the needle. This narrowing of the foramen of Monro is brought about by edema or a displacement as the result of a tumor.

The danger of emptying too rapidly is that it produces headache, nausea and vomiting, which is avoided by the gradual emptying.

All of our work has been done under local anesthesia in infants and children up to 9 years of age, as well as in adults. (Showed series of slides demonstrating different cerebral lesions.)

Towne of California introduced the taking of four additional views, using the horizontal ray which indicates the fluid levels as well as the proper change in the location of air which results when the position of the head is changed. These additional views add to the composite and aid in arriving at a definite roentgen-ray interpretation.

The communicating types of hydrocephalus are difficult to treat; here the intra-abdominal drainage has been attempted but reported more or less of a failure.

Indications for cerebral pneumography are: 1. In cases where a tumor cannot be localized by other means. 2. In cases operated on and no tumor found. 3. In cases which exhibit secondary manifestations so pronounced as to completely overshadow the early clinical symptoms which result from general pressure phenomena, displacement or edema. 4. As a corroborative procedure when it is necessary owing to the fact that the clinical history, the examination of the patient and observation has not been sufficiently conclusive to aid in the localization of the tumor. 5. To gain information as to the size and extent of the tumor. 6. In cases of hydrocephalus in determining the site or location of the structure which causes this accumulation of fluid. 7. In localization of the origin of a tumor, as in the case of brain tumor existing for some time suddenly developing Jacksonian epilepsy, which may result from either extension, pressure, hemorrhage or edema and yet the tumor may originate in the extreme frontal region, subcortical or parietal areas. This permits of the opening of the skull directly over the tumor rather than a wide opening over the motor region, often far removed from the tumor. He believes ventriculography is another method added to our already well known methods of diagnosis of intracranial lesions and urges its use as a laboratory method of procedure and not as a direct method of diagnosis.

QUESTION: What is the direction of the needle?

DR. DAVENPORT: We enter at Kocher's point on the right side, the point of the needle directed slightly backward and inward, going in about from 4 to 5 cm. in the average case. In three instances we have struck the tumor with the needle, when we promptly entered on the other side in order to verify these facts.

QUESTION: Have you seen any untoward results appear suddenly?

DR. DAVENPORT: No case has been lost from the ventriculography procedure. Some patients have had nausea and vomiting but this usually passed off promptly, providing one replaced the fluid by air and did not resort to inflation of the ventricles.

A Report on Typhoid in Children. Presented by Dr. Maurice Blatt.

We have six cases of typhoid in the Children's Hospital today. The last patient was admitted about two weeks ago. This is a decided increase over the number of cases of typhoid in the city last year. The last patient admitted to the Children's Ward of the County Hospital last year was on October 9. Although this is November, we are still admitting patients with typhoid, and there are five or six cases reported in the city each day. The location of the epidemic is, so far as I am aware, principally on the south side. Every effort is being made to trace the infection. The water and crib have been thoroughly examined and the mains are perfect, so oysters and fish are under suspicion. There were 230 cases last year, of which 23.5 per cent. were in children, showing the importance of the subject from the pediatric standpoint. Thirty persons died from typhoid.

Last year we had nine cases of typhoid during the summer, beginning March 20, and ending October 9. This year we have had so far twenty-two cases of typhoid representing the same group, and the cases are still coming in. Those of you who remember back as far as I do will remember when typhoid was considered a serious disease in Chicago, and when recruits were asked from medical schools to come and do night nursing in the typhoid wards. He then spoke of the use of vaccine. We have a family in the hospital, with two cases out of six, in which all the other members were immediately protected against typhoid fever. The child who developed the fever second had received only the second dose when the disease developed. It does not seem to take as long to produce protection in typhoid as it does with the toxin-antitoxin. In these cases hemorrhage and perforation are rare, but in the last two years one case of hemorrhage occurred, and we have here a child with a number of relapses, to which I wish to call your attention.

The histories of these children, obtained from their parents, are almost identical. All complain of anorexia, of malaise and some diarrhea; occasionally they complain of chilliness, rarely of cough; last year there were some with nose bleed but so far this year there has been none. Sometimes a chilliness, but always the malaise, is the dominant feature attracting the parents' notice, Absolute diagnosis is dependent on two things, the leukopenia and the Widal test. Blood cultures have not been very successful but the Widal test is universally successful and with a very high titer in many cases. As corroborative evidence of typhoid incidence we have the fecal and urinary cultures, which always come back positive.

The length of stay in the hospital has averaged about four and a half weeks. Some were forced cases which had almost reached the convalescent stage when they were brought to the hospital, and were only in from twelve to fourteen days when the cultures became negative and they were sent out. Most cases come in at the beginning of the second week, so probably the average length of the disease is about six weeks. The history of one case has been very interesting.

L., a girl, aged 6 years, came into the hospital Oct. 19, 1923, with a history of cough, fever and diarrhea of two weeks' duration. Her stools had been liquid yellow, she had abdominal pain for one day and considerable malaise. The onset had been abrupt, with a chill. Examination showed numerous harsh râles in both lungs; a palpable spleen, and a white count of 4,200 with 2,720,000 reds. On October 22 the Widal test was positive, 1 to 1,280, and on the following day typhoid organisms were recovered from the stool and urine. Her tuberculin reaction was negative. A blood count ten days after entrance showed a white count of 4,800. The temperature curve, which is similar to that seen in the majority of our cases, was as follows: The first week showed 100 F. at the low point and 104.5 at the high point. The second week varied between 103 and 98. The third week ranged from 100 to 98. During the fourth week 100 F. was reached only once, the temperature remaining fairly constantly below

99. The diurnal range was a characteristic low point in the morning and high in the afternoon.

Most of the children do not come in this year with a history of bronchial irritation, as was the case last year. Many of the patients last year had to be differentiated from miliary tuberculosis or acute bronchitis with temperature.

The following is the history of another child whose case was rather puzzling:

A girl, aged 8 years, came into the hospital on June 23, 1923. She had a history of a temperature for one week but was not in good health the week before. There was loss of appetite, drowsiness, and a septic type of temperature from the beginning. Physical examination showed moist râles and a few subcrepitant and sonorous râles. Slight rigidity of the abdomen was present, accompanied by moderate tenderness. The first blood examination showed a white count of 7,500, 44 per cent. of which were lymphocytes, and a negative Pirquet test. The Widal test was positive. On June 26, three days after admission, the white count was 8,800. On July 10, typhoid organisms were obtained from the stool. The temperature during this time had a high range up to 104 F. during the first week. In the second week the temperature was 103 with a low point of 100. In the third week it did not drop below 102 at any time, and in the fourth week it ranged between 102 and 105. At this time the child had a moderate intestinal hemorrhage, with a poor pulse ranging from 160 to 180. The temperature dropped to 100 with this hemorrhage but rose to 102 the following day. Prior to this hemorrhage the pulse range was between 130 and 140. This was on July 19. On the following day there was a second hemorrhage, and the white blood count was 7,800, the hemoglobin 45 per cent., and the coagulation time two and one-half minutes. In the sixth week the temperature ranged below 99. In the seventh week there was a relapse, with the temperature reaching 105, with sharp peaks and drops to 100. The eighth week the curve ranged from 102 to 98, gradually falling to normal, when in the eleventh week the child was discharged well.

He presented a temperature chart of one patient with a prolonged temperature. The patient was still in the hospital, then in his tenth week. There had been several relapses but no other complications. The case resembled pulmonary tuberculosis, not only because of the constancy of the temperature with its rise and fall, but because there was very little swing in the temperature for some time and there were moist râles in the chest. At the time of the second relapse the temperature reached 105 and in the twelfth week the child had a temperature of 98 for the first time.

Regular Meeting, Dec. 18, 1923

H. W. CHENEY, M.D., *President*

CONGENITAL ABSENCE OF THE RADIUS. Presented by DR. KATSUJI KATO (by invitation).

Congenital absence of the radius was once thought to be rare, but there are 250 recorded cases, showing approximately an equal occurrence of bilateral and unilateral defect. The absence of the bone is usually complete, only 12 per cent. being incomplete or rudimentary. The principal pathologic change is the absence of the radius which is of congenital origin, and the ulna is usually hypertrophied and curved, while the hand shows radiopalmar deviation. The absence of the thumb is rather a constant finding, while syndactylism and polydactylism are frequently associated. The associated pathology also includes one or more other congenital anomalies.

In this paper, three cases are reported from the orthopedic service of Dr. Edwin W. Ryerson, the Children's Memorial Hospital, Chicago.

CASE 1.—J. P., a boy, Italian, showed complete bilateral absence of the radius. The left thumb was normal but the right attached loosely to the hand by means of a fibrous tissue and fell off two months later. The child finally succumbed to pneumonia at the age of 2 years.

CASE 2.—F. K., a girl, American, was first seen in the Dispensary at the age of 2 months when the diagnosis of congenital absence of the right radius was made, on the strength of characteristic radiopalmar clubhand and transposition of the radial artery, which was later confirmed by roentgenologic evidences. At 6 years of age, Dr. Ryerson performed the operation on the affected limb when a bone graft was taken from the hypertrophied ulna and inserted into the radial site, the forearm subsequently being held in extension by a plaster cast.

CASE 3.—B. S., a girl, American, showed partial absence of the radius, the upper fragment of the bone being normal. The later history of the patient is lacking as she failed to return for further observation and treatment.

The etiologic consideration of the defect is interesting although more or less theoretical. Of the more important theories, Gegenbauer's archipterygeal theory, Dareste's amniotic compression theory are worthy of thought, but none of them appear to be adequate. The author advances a theory of embryonic cellular dystrophy, which considers the defect as arising from cell anomalies which might have resulted from various factors, such as inherent vagaries of chromosome material, defective cytoplasm, trophic nerve disturbance, abnormal blood supply or some form of fetal or maternal inflammation, producing a veritable scar tissue which inhibits the normal development of the osteogenetic mesoderm.

The treatment is largely surgical. The prognosis is gloomy. In many cases the unfortunate victims die young because of the seriousness of associated anomalies. When they survive till childhood, they become an easy prey of intercurrent infection. The correction of the deformity depends on the degree of pathologic changes, but it can usually be remedied, although the deformed hand remains always weak and the affected extremity always atrophied.

This presentation was discussed by Drs. Grulee, Brennemann and Cheney.

THE INTRADERMAL SALT SOLUTION TEST. II. ITS PROGNOSTIC VALUE IN CASES OF SO-CALLED NEPHRITIS WITH GENERALIZED EDEMA. Presented by DRS. C. A. ALDRICH and W. B. MCCLURE.

In July 1923, a preliminary report on observations on the time required for the disappearance of intradermally injected salt solution was published. It was found that the elevation produced by the intracutaneous injection of 0.8 per cent. sodium chlorid solution disappeared much more rapidly in cases with edema than in a small group of controls. The technic is as follows:

Two-tenths cubic centimeter of an 0.8 per cent. aqueous solution of sodium chlorid is injected intracutaneously under aseptic conditions. Duplicate injections are made about 2 cm. apart, either in the forearm or in the leg, or in both. In the forearm the flexor surface is used, and in the leg, either the inner surface of the calf or the lateral side of the anterior surface of the leg. The needle is inserted sufficiently superficially so that the lumen is visible through the skin. The end point of the disappearance of the elevation can best be determined by palpation unassisted by inspection, since color changes seen about the point of injection may cause confusion. It is necessary to disregard the very small traumatic elevation that is occasionally produced just where the needle enters the skin. This may persist after the large elevation, due to the injected salt solution, has disappeared.

We have come to consider sixty minutes as a lower limit of the normal range.

This paper reports the observations made on fifteen cases with generalized edema and pathologic urinary findings without evidence of cardiovascular disease.

A summary of the observations shows:

1. In a general way the greater the edema the shorter was the absorption time and vice versa.

2. In all cases, where tests were made previous to the occurrence of edema, demonstrable by pitting, there was a reduction of the absorption time, days and sometimes even weeks, before pitting was demonstrable. The absorption time in improving edematous cases was increased before the edema showed any apparent decrease, in all cases tested at such opportune time. During improvement it has closely approached the lower normal limits before the pitting was entirely gone.

3. There was no constant relationship between the absorption time and the severity of the urinary findings.

4. There was a marked parallelism between the absorption time and the urinary output, although the change in the absorption time usually preceded the change shown on the urinary output chart.

5. Recovery sometimes occurred even when the absorption time was less than one minute. However, in every fatal case the absorption time in the leg fell below one minute.

6. During improvement with weight gain, the absorption time curve tended to parallel that of the weight. During changes of weight due to retention or loss of water, these curves were in opposite directions.

7. In three cases tested only during or after convalescence, the time was within the normal range.

Graphic charts of observations on five of these cases were shown, demonstrating the points given above.

Charts of two cases with cardiovascular disease, pathologic urinary findings and edema were shown in contrast.

We believe these results justify the following conclusions:

1. This test is a valuable means of determining the immediate prognosis in such cases, and shows changes at an earlier date than shown by any other means with which we are familiar.

2. It is an aid in directing the therapeutic management of these cases.

3. These results are confirmatory evidence in favor of the theories that the tissues in this type of case are active in the development of edema.

4. It may be of considerable value in differentiating between circulatory and "renal" types of edema.

DISCUSSION

DR. W. B. MCCLURE: Dr. Aldrich mentioned the fact that we had found decreased absorption time after ether anesthesia. I wish to speak more in detail of our results in the study of these cases.

In a series of ten children receiving ether for surgical procedures, principally tonsil and adenoid operations, we found a reduction of the absorption time ranging from 81 to 24 per cent. following the anesthesia. This effect did not differ materially in the cases in which atropin was used and those in which it was not used. It is known that ether anesthesia produces a certain degree of acidosis. This acidosis may logically be the cause of the decreased absorption time, although the possibility of the participation of other toxic influences, due to the anesthesia, must be considered. This is of interest in connection with the case referred to by Dr. Aldrich where the tonsillectomy was done, through an error in interpretation of orders, at a time when slight pitting and

a drop in the absorption time had indicated a beginning edema. The child developed a very marked edema following the anesthesia, which we thought might have been due, in part, to the anesthetic.

Dr. Jesse R. Gerstley: As I understand, the technic of the test is 0.2 of 1 c.c. May I ask just how accurately this must be measured? I should think that a difference of a few tenths of a cubic centimeter might not be very important in this connection.

Dr. Aldrich (in closing): As regards the amount of the solution injected, I do not think it makes a bit of difference exactly how much you use. As a matter of fact, in doing this test in some of the cases the needle, or syringe, leaks while we are injecting the solution and we find that one wheal is larger than the other when it is put in, but that they both disappear at the same time. Theoretically, it would not make any difference and practically we have found that to be true, but if one is to give any idea of the test it is necessary to have some knowledge of the approximate amount we are using.

Regarding the use of alkalis, I think many of their urines were not alkaline at the time of testing. We have put most of the patients on alkalis, however. In the number of things to be done that is one of the things that has not yet been investigated thoroughly, but it should be. Also, the question of salt intake and output should be studied. We have done one test recently to see whether various concentrations of salt would make any difference, figuring that if osmotic influences were predominating in edema, as some claim, if we put into the skin a solution of two or three times the salt concentration normally found in the body, the fluid should remain a long time in the skin. The disappearance of the wheals showed a difference of only one minute or so. We used three different strengths of salt, one 1.6, another 2.4, and another 3.2, the last being in excess of any concentration ever found in the body. That solution was absorbed almost as rapidly as the others. While that needs to be confirmed, I think it shows that the osmotic factors are not the predominant ones at work.

When we did this test at first it took a long time to figure out how long the wheal normally lasted. Because of some experiments on rabbits we thought that it disappeared within a few minutes. We kept on working and found sixty minutes which at first we thought was pretty high, but which we now consider a comparatively short time. The higher the time the better for the patient. We go to the patient without knowing where we have put the injection and if when we feel the arm or leg we discover the location of the wheal we know it is still present.

Regular Meeting, Feb. 19, 1924

H. W. Cheney, M.D., *President*

Concerning the Etiology of Hypertrophic Pyloric Stenosis. Presented by Dr. Louis W. Sauer, this issue p. 608.

DISCUSSION

Dr. Joseph Brenneman: I want to ask one question. Some years ago Hess of New York made a statement which in my opinion was not especially confirmed by evidence which he presented, that with a pyloric spasm there were other spasms in the intestinal tract, namely, cardiospasm, rectal sphincter spasm, and general hypertonicity of the whole intestinal tract. I wonder if that idea has received support elsewhere. There are two other conditions which I think closely simulate pylorospasm. I saw one such case in a child aged 2 weeks. The child had typical peristaltic waves, except that they did not point quite right, they pointed toward the navel instead of the liver. This

proved to be a case of volvulus. The other condition, which I have seen twice, was duodenal obstruction. In each case the baby vomited quantities of bile, and this of course distinguished it from pyloric obstruction, in which this could not occur.

DR. CLIFFORD GRULEE: Greenebaum observed a child operated on, aged 2 days. That would seem to be a case where it was a typical pyloric stenosis, and it would support the theory that it could be a congenital anomaly. Another condition associated with vomiting and also frequent is rumination. I have seen in this many times active peristalsis, in cases which were not true pyloric stenosis in the ordinary sense of the word.

DR. JULIUS H. HESS: Has Dr. Sauer had occasion to study any fetal cases? I have sections of pyloruses taken from fetuses ranging from 18 to 28 weeks. All show a very well developed layer of circular and a thin longitudinal muscle, but even more striking is the marked infolding of the mucosa. In the fetus, the mucus membrane of the pylorus is very abundant and appears excessive when compared with cross section of older infants.

Is it not possible that the consequent infolding and compression of the mucosa may not act in such a manner as to cause repeated contraction of the circular muscle fibers resulting in gradual hypertrophy either in fetal or early extra uterine life?

DR. SAUER (closing): I came across several articles in the literature which speak about this spasm of the entire intestinal canal. I spoke to Dr. Alvarex, who has written extensively on the intestinal canal, and he thought the condition was simply spastic. Professor Carlson thought if we studied the condition we would not find hypertrophy. We read so much about pyloric spasm, especially in American literature, but in the last few years Holt said the thing does not exist. He said these are mild and severe cases of hypertrophy.

Mohr has had two cases, which he published, of pure pyloric spasm which ran a long while and ended fatally, without hypertonia. He published a report on one of them, which he had followed for weeks, in great detail. Greenebaum had a typical case, operated on the second day. With regard to the fetal pylorus I have not done much, but a number of Danish and Norwegian anatomists have published wonderful works on this subject. The only ones who disagree with this work, that is, that the pylorus in the fetus may show a marked stenosis, are the English physicians. Cunningham and one or two others claim the muscle is thicker a few weeks after birth than during fetal life. But most of the work shows very defintely that in the fetus the muscularis is distinctly thicker.

CELL COUNTS IN BLOODY SPINAL FLUID (INAUGURAL THESIS). H. E. IRISH.

A detailed technic is presented for counting white cells in slightly bloody cerebrospinal fluid. Test counts are reported on ten fluids made by three counters for checking against each other and against a computed number of cells. The results shown are approximately correct in average from the standpoint of diagnostic usability. It is shown that accuracy was increased by promptness in counting. The use of this same technic by three counters in ten fluids showed an increase in accuracy of 133 per cent. in white blood cells and 8 per cent. in red blood cells, over five fluids counted by the two counters using their usual methods.

Slightly bloody fluids are shown to be usable diagnostically. The use of this technic where much blood is present or in purulent fluids is not discussed in this paper. Purulent fluids readily reveal their identity by their great increase in white cell content. Bloody spinal fluids are best counted immediately, but can be transported and counted within a few hours with approximate accuracy.

The technic used to count leukocytes in spinal fluid was as follows:

1. Draw stain (Wright's methylene blue 50 per cent., glacial acetic acid 50 per cent.) to Point 1 in white pipet.
2. Shake fluid thoroughly and draw up from its central portion to Point 11.
3. Rotate and shake pipet for one minute.
4. Let stand ten minutes for complete staining and decolorizing.
5. Shake again thoroughly, allow first few drops to escape from tube and then place drop in counting chamber.
6. Use low power to find counting lines, then high power to ascertain the effect of stain.
7. If whites are fully stained and reds are completely decolorized, use low power for counting.
8. If contrary is found, use high power for count.
9. Focus up and down on doubtfuls. Increased refractility decides identity of whites.
10. Count two full fields of 9 large squares (Levy ruling) on 16 (Thoma Zeiss).
11. Take average of two fields and multiply by 10/8.

To count erythrocytes in spinal fluid:

1. Shake fluid thoroughly, use drop from central portion to place on counting chamber.
2. Count 5 large squares (= 80 small squares) and multiply by 0.50.
3. If cells are too numerous to count in chamber, dilute with Hayem's solution and use proper multiplier.
4. The results of these tests are shown in tables.

DISCUSSION

Dr. Clifford G. Grulee pointed to the practical application of this paper.

Dr. Ray L. Critchfield: Recently at the Children's Hospital we have changed our method of collecting spinal fluid for the white cell count.

We now place 1 mm. of glacial acetic acid in a test tube and permit 9 drops of spinal fluid to fall into the tube directly from the needle. We find that the fluid stands up better and this method does not interfere with the accuracy of the count. All counts must be made within fifteen minutes from the time they are obtained, which we believe makes for greater accuracy.

Dr. Irish (in closing): Answering Dr. Grulee, the range of utility of the technic probably will be limited to fluids that have a cell count varying from 50 to 100. In that particular range of cell counts are tuberculosis, poliomyelitis, one form of syphilis, mild septic meningitis and the like. In this field, very often the number of cells in the spinal fluid is of great service in indicating the nature of the fluid. The increase would have to be calculated with great care in order to avoid being misled by it. In counts over 500 the white cells are usually sufficiently numerous so that a smear will show the nature of the fluid.

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